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CONGENITAL ATRESIA OF THE ŒSOPHAGUS; AN OPERATION DESIGNED FOR ITS CURE

WITH A REPORT OF TWO CASES OPERATED UPON BY THE AUTHOR

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OF the various types of congenital malformation of the œsophagus, atresia, at about the level of the bifurcation of the trachea, with a tracheo-œsophageal fistula, is by far the most frequently found, forming seventy per cent of all cases. It is to the treatment of this type only that my paper is directed.

Less than 150 cases of congenital atresia of the œsophagus have been reported to date, but cases are being recognized with increasing frequency, and, stimulated by our advance in knowledge and results of treatment of congenital pyloric stenosis, the subject bids fair to assume a place of importance in the surgery of infancy.

The lesion is essentially fatal when untreated, and to date treatment has been unavailing. The present paper presents a plan of operation the principles of which were carried out in two cases personally operated upon, and which seems to me to offer some hope of success.

PATHOLOGICAL ANATOMY OF ŒSOPHAGEAL ATRESIA

The site of the atresia is on a level with the bifurcation of the trachea.[1] There is a complete separation of the two segments of the gullet. The upper segment ends below as a dilated blind pouch, behind and just above the tracheal bifurcation. The lower segment joins the stomach at the cardia in the usual manner. The *essentially fatal* factor in the anatomy consists in a communication between the upper end of the distal segment and the trachea or bronchus, allowing of free communication between lungs, gullet, and stomach; forced respiration, as in crying, etc., distends the stomach; attempts at vomiting fill the air passages with stomach contents (secretion), resulting in drowning (cyanosis).

In a small proportion of the cases reported, the connection between gullet and trachea did not exist; here a simple gastrostomy would suffice to maintain life; but this favorable condition is rare. As in other congenital anomalies, accompanying malformations are not infrequent. Case 1 showed a horseshoe kidney. Dr. Brennemann (5) reports a case in which gross ureteral changes were found, consisting of a doubling of both ureters. There were also microscopic changes in the spinal cord. According to Brennemann, one-third to one-half of the reported cases show accompanying malformations.

Symptoms. All four of my cases were grossly well-formed, fully grown infants. Contrasting strongly with my experience in congenital pyloric stenosis, all showed symp-

[1] For a more complete account of pathological anatomy, and theories as to embryological origin, see Zeit (1), Shukowsky and Baron (2), Konopacki (3), and Hirschsprung (4).

toms beginning immediately after birth. These may be summarized as follows:

1. Everything taken regurgitated at once, beginning with the first attempt at feeding.

2. Continuous discharge of saliva and mucous from mouth.

3. Spells of cyanosis at frequent intervals, especially brought on by feeding.

4. The abdomen distended by crying and coughing.

5. Difficult respiration; marked inspiratory retraction of chest and abdomen.

6. Passing catheter into œsophagus discloses a complete block 10 to 12 centimeters from gums (at bifurcation of trachea).

7. An early rise of temperature, with cough, usually indicates the development of pulmonary involvement from the constant regurgitation of fluids from below. This will prove a serious handicap to any method of surgical treatment that otherwise might prove successful. The swallowed tracheal secretion, accumulating in the proximal segment of the gullet, early becomes purulent. As stated elsewhere, a smear of this fluid in Case 4, obtained by aspirating with catheter and syringe, was purulent, with many cocci.

The attacks of cyanosis are due to regurgitation of fluids from the stomach. This would not account for attacks precipitated by being fed. Probably disturbed intrathoracic tension caused by the tracheo-œsophageal communication prevents the protection of the upper end of air passages from the entrance of fluids.

The difficult respiration is probably associated with early development of broncho-pneumonia, and with blocking of the lower end of the trachea.

The symptom complex has been repeatedly described. It is that of tracheal obstruction, broncho-pneumonia, and starvation.

Operative Treatment. Operative treatment heretofore carried out has been gastrostomy or jejunostomy. Hoffmann (6) attempted to close the gullet, but desisted, and did a gastrostomy. His attempt was by way of the neck, an obviously impossible route. In all cases, feeding through the new opening has been followed by immediate or ultimate death by drowning, or the production of such marked dyspnœic or coughing attacks as to forbid persisting in the attempt. In Brennemann's first case, a jejunostomy was done by A. E. Halstead; apparently no regurgitation occurred on feeding, but it seemed impossible to keep the child alive by administering its food per jejunum. Jejunostomy cannot, of course, obviate the fatal outcome of the tracheo-œsophageal fistula.

Direct anastomosis of the ends, with closure of the trachea, in an infant less than a week old is certainly a hazardous proceeding. I am not certain, however, that it may not prove possible, and, if so, would be the ideal operation. I do not wish to dismiss the idea of immediate union of the two segments of the œsophagus. The technical difficulties would be rather greater than that of the operation here suggested. The greatest obstacle would be the danger of infection; however, I expect to attempt it at the first favorable opportunity. The advantage of the operation described below lies in the fact that in dividing the infected fistula one can crush and invert the structures with a minimum of soiling. The present problem seems to be to close the communication with the trachea, do a gastrostomy, and so feed the child. The recent development of thoracic surgery justifies the hope that later in life a means of utilizing the gullet may be found.

PLAN OF OPERATION

I. Closure of gullet at tracheal junction by trans-thoracic route.

II. Gastrostomy.

Operation:

I. *Closure of Gullet:* Closure must be at the junction with trachea.

1. No pouch should be left to accumulate fluid, which must quickly become infected, and be a constant source of danger.

2. No part of the œsophagus must be sacrificed; it must be kept full length, to be utilized later if restoration of the canal seems feasible.

The possibility of saving the infant centers about the œsophagus. In the two operations carried out by the author, the thorax was opened widely under positive intrapulmonary tension, the gullet isolated at its junction with

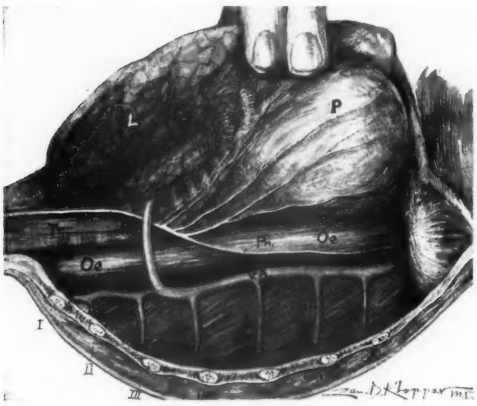

Fig. 1. Normal structures as seen with lung drawn forward and medianward. L, lung; P, pericardium; Tr, trachea; Oe, œsophagus; Pn, pneumogastric nerve; V. A., vena azygos; I, II, III, etc., ribs.

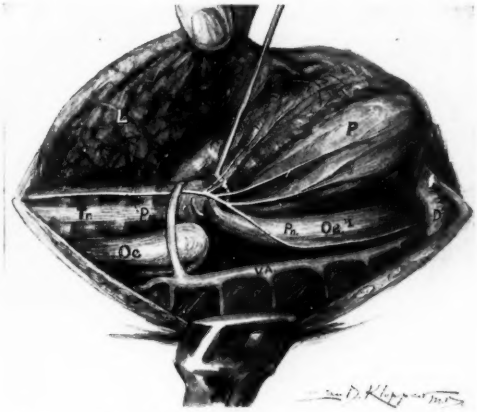

Fig. 2. View of malformation and adjacent structures, as seen with lung drawn forward and medianward. Note complete separation of upper and lower segments of œsophagus and relation of pneumogastric plexus to tracheo-œsophageal junction. Lettering as in Fig. 1.

the trachea, in both cases at the point of bifurcation of the latter, and a ligature passed around it. The expectation was that the ligature in cutting its way through would allow of cicatrization and closure of the tube above and below. The possibility of the tube being re-established was recognized, but it must be remembered that in spite of this tendency, it is the exception and not the rule for the lumen of a tube lined with epithelium to re-establish itself; e. g., sterility is not assured by ligation of the tubes, but is rendered extremely probable. The objection to dividing the structure and closing in the openings by means of sutures was the danger of infection; this is, however, the more logical procedure, and should, I believe, be carried out. Lack of familiarity with the surgical anatomy of the parts will obviously be a source of embarrassment to most general surgeons. It was to the author. The smallness of the parts in a new-born infant and the peculiarly difficult site of operation made the hazard apparently insuperable. A rather simple procedure served materially to lessen the difficulty. The abdomen was first opened, a small opening made in the anterior wall of the stomach at the intended site of the gastrostomy, and a pliable uterine sound introduced, passed up through the cardia into the gullet. Leakage was prevented by a circular suture. The child was now turned over and the gullet cut down upon and found without difficulty.

Technique:

1. Protection from shock, loss of heat, etc. Extremities well wrapped in cotton batting or woolen bandages. Warm water bags, etc.

2. General ether anæsthesia. Intratracheal ether anæsthesia under a positive pressure of 6 to 8 mm. of mercury during the thoracic part of the operation, with 12 to 15 mm. pressure before inserting the final suture, to bring the lung well up to the chest wall.

My apparatus consists of a motor and pump, giving a free current of air at a pressure varied readily by a rheostat, with a valve in the connecting tubing, through which excess air may be allowed to escape. The air passes over ether contained in a Wolff bottle, or is shunted directly to the tracheal tube as desired. A manometer and a mercury "blow-off" as a safety valve are in circuit. The whole is simply the type of apparatus of Meltzer and Auer. Just before beginning differential pressure, *and not until the operator is ready to begin it,* a pad should be firmly bandaged upon the abdomen; otherwise the stomach and intestine will be greatly distended by inflation through the tracheo-œso-

phageal fistula. On the other hand, the pad must not be applied until the tracheal catheter is ready to be inserted, or until *after* it is inserted, lest the child be drowned by forcing stomach contents up into the lungs.

In my cases, having opened the abdomen to insert the œsophageal sound from below before doing the thoracic part of the operation, I simply clamped the jejunum; but I believe I shall find the preliminary laparotomy unnecessary in future work.

3. Incision in sixth right interspace, beginning at about the angle of the ribs behind and extending forward approximately two inches. The ribs are widely separated and a finger inserted to keep the inflated lungs away. The posterior end of the incision is now carried vertically upward over three or four ribs, the latter divided with strong scissors. By drawing the ribs upward with a little force as they are divided, little blood will be lost, and no artery clamps will be found necessary until this step of the operation is completed. They may now be applied to the divided intercostals and such other bleeders as may appear, though a little pressure with dry gauze, kept up for a few moments, will probably suffice. These minutiæ may appear trivial, but this stage of the operation should be completed in less than five minutes, and the entire operation must be carried out quickly if the operation is to be at all feasible.

It will be noticed that access is obtained through the *right* side. The normal œsophagus crosses the *left* bronchus. In both of my cases the gullet was apparently to the right of the median line. In my first case I went through the left side and found much more difficulty than in my second case.

4. The ribs are widely spread, and a flat retractor used to draw the lungs forward and medianward. The gullet can readily be made out, and only the extreme importance of the adjacent structures renders the next step of the operation tedious and difficult.

5. Isolation of the gullet. In my cases I first intubated the gullet from below, as previously described, and I should advise this procedure to anyone attempting his first case. With the experience of two cases and the dissection of a number of infant bodies, I believe I shall find this unnecessary in the future.

The structures in the field of operation are readily defined. The gullet stands out prominently and dips acutely forward to join the trachea. The pneumogastric nerve is plainly visible. It comes down from above in a single trunk, lies behind the azygos vein just as the latter turns in to meet the vena cava. Just below the azygos, anterior and dextrad to the tracheo-œsophageal junction, it divides into numerous branches much after the manner of the pes anserina of the facial nerve. Viewing the parts as seen during the operation, with the lung retracted, the plexus so formed lies to the right, the ascending part of the azygos to the left and the transverse part of the azygos above the field of operation. From the pneumogastric plexus, and made up of it in part, the main trunk of the pneumogastric dips down to the side of the gullet, just below where the latter turns to meet the trachea. In the dissection of the specimen from Case 3, it was found that the vagi were not included in the ligature. As the vagi lie in but loose contact with the gullet, the proximal one in plain sight, and are readily moved by drawing on the reflection of the pleura, I feel safe in stating that they can always be avoided.

The lower segment can be followed to its junction with the trachea. The latter can be palpated to help orientate oneself. A blunt hook is passed around the gullet. No bleeding was encountered in either of my cases, but it is evident that manipulation must be gentle or troublesome bleeding may readily occur.

6. Closure of the gullet. Ligation was used in both of my cases. A more definite closure, with separation of the parts, is desirable. The part should be crushed with a heavy clamp, the crushed tissue cut, and both ends turned in. This is really the crux of the whole operation. All other steps of the operation are absolutely dependable. One may hesitate before attempting so drastic a procedure as a major laparotomy and thoracotomy in an infant three or four days old; but my second case was awake and out of shock shortly after the operation was completed. The shock can, therefore, be withstood. Closure of the gullet

means, however, cutting through infected tissue in a part most difficult to protect from infection. Possibly some advantage might be gained by using the Downes type of thermoclamp, and cutting through the cauterized tissue. In either case, the open ends of both gullet and trachea must be invaginated, the latter as carefully as the former, or it may be torn open by the differential tension used.

Closure of Chest. The chest is rapidly closed. A single pericostal suture (Sauerbruch), applied first, will bring the sixth and seventh ribs together, and a few deep sutures of the soft tissues of the longitudinal part of the incision will practically close the cavity. Before inserting the last suture or two, the intrapulmonary tension should be raised to about 15 mm., which will bring the lung well up to the chest wall.

II. *Gastrostomy.* The gastrostomy is made in the usual manner, of the type preferred. When a sound is inserted as a preliminary step in the operation, the site should be chosen so as to utilize the opening made.

REPORT OF CASES

I have had four cases of congenital atresia of the œsophagus. Two of these have been previously reported, by Dr. F. R. Zeit, from the pathologist's viewpoint, and by Dr. J. W. Brennemann, from the clinical standpoint. In the first of these I did a gastrostomy, only to have the child almost die by drowning upon receiving its first food. The second case I allowed to die without interference, feeling intervention to be hopeless. The third and fourth cases, not previously reported, were both subjected to the operation here described. Case 3 died of shock shortly after operation. Case 4 lived twenty hours. It began to awaken immediately after leaving the operating room and reacted fairly well from shock within two hours. The violent respiratory efforts ceased immediately after operation, the breathing becoming normal though rapid. Beginning immediately upon the completion of the operation, it was given fluids per gastrostomy tube every half hour, receiving a total of 8 ounces, without having a choking or "cyanotic" spell. Some bleeding from the laparotomy incision occurred, but not sufficient externally to be of importance. In both cases oscultation immediately after operation showed the lungs to be in contact with the chest wall.

Case 3 obviously died of shock.

Case 4 had a purulent bronchitis or broncho-pneumonia before beginning operation. There was also a purulent secretion in the proximal blind gullet, which was aspirated with a catheter. Smears of this showed richly cellular pus, with many cocci, grouped as diplococci and staphylococci.

The cases which I wish to add to the casuistics of congenital atresia of the œsophagus follow. The symptoms and pathological findings were essentially those as given, and will not be repeated:

Baby E., male, second child, weight 7 pounds, 8 ounces. Case No. 42992, Wesley Hospital, July 16, 1913. Referred by Dr. Sandstedt of Chicago.

Operation. Preliminary gastrostomy under ether. Intubation of lower segment of gullet from below with metal sound. Change to positive pressure ether anæsthesia. Incision in left seventh intercostal space, carried upward from posterior end. Five ribs divided near their angles. A silk ligature carried around the gullet, using the sound as a guide. Seventh and eighth ribs brought together by a suture that included both (Sauerbruch). Intrapulmonary tension raised until lung came to chest wall, when remainder of wound was closed. Gastrostomy of the E. J. Senn type now completed and water immediately given through tube. Auscultation showed lung in contact with chest wall. The operation was unduly prolonged, and the technique faulty in that the incision was made on the left side and too low. Death occurred in two hours, water having been given through gastrostomy tube three times without evidence of entrance to lungs. Postmortem showed the fistula type of anomaly, with the ligature placed at site intended. Lungs fully inflated. No blood or fluid in either chest or abdomen. No gross accompanying malformations. No evidence of opposite pleura being opened during manipulations.

Baby B. Case No. 73248, Presbyterian Hospital, August 24, 1913. Born in the obstetrical service and transferred to Drs. C. G. Grulee of the pediatric and Carl Davis of the surgical departments, to whom I am indebted for the privilege of operating upon the infant. Male, of Russian Hebrew parentage, 3 days old. Weight at birth, 8 pounds, 6 ounces; temperature, 98.6° F. Mother a multipara. Child had a marked purulent secretion in its mouth and gullet, and showed marked respiratory retraction of ribs and abdomen.

Operation. Preliminary intubation of gullet from below, as in previous case. Clamp placed on jejunum to prevent inflation of bowels. Temporary closure of abdominal incision. Change to intratracheal ether anæsthesia. Incision in right sixth intercostal space about two inches long, the posterior end carried upward and the sixth, fifth and fourth ribs cut across near their angles. Retraction of ribs gave perfect view of field of operation. A blunt hook passed around gullet at tracheal junction and ligature placed. Chest closed as before, raising the intrapulmonary tension sufficiently to bring the lung up to the chest wall. Gastrostomy of the E. J. Senn type.

The child took approximately 8 ounces of fluid through the gastrostomy tube in next twenty hours without showing any evidence of regurgitation into lungs. Its breathing lost the dyspnœic type at once. No attacks recurred. Reaction rapid. Auscultation showed lung maintained in fully inflated condition. Death 20 hours later. No post-mortem examination permitted.

REFERENCES

1. Zeit, F. R. J. Med. Research, 1912–1913, xxvii, 45–54.
2. Shukowsky and Baron. Arch. f. Kinderh., 1912, lviii, 191–198.
3. Konopacki. Zentralbl. f. allg. Path. u. path. Anat., 1912, xxiii, 386–394.
4. Hirschsprung. Hospitalstidende, 1895, xlii.
5. Brennemann. Am. J. Dis. Children, 1913, v, 143–150.
6. Hoffmann. Diss., Greifswald, 1899. Referred to by Griffith and Lavenson, Arch. Pediat., xxvi, 3, 161.

THE RÔLE OF THE GLANDS OF INTERNAL SECRETION IN THE GENESIS OF FIBROMA MOLLUSCUM GRAVIDARUM [1]

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IN 1906 I presented a paper [2] before the New York Academy of Medicine, in which I described as a new clinical entity a lesion of the skin originating during the later months of pregnancy and which I named "Fibroma Molluscum Gravidarum." So many changes have taken place in my conception, particularly as to the etiology and the course of the condition, that the present occasion seems an appropriate one to offer my modified views.

Terminology. The name, fibroma molluscum gravidarum, was chosen because the lesions correspond pathologically with the disease known and described as fibroma molluscum by dermatologists. The clinical course, however, is widely different, and as the lesion occurs only in pregnant women, the modifying word was added. The sole similarity between the ordinary fibroma molluscum and that of the lesion under discussion is in the microscopic picture.

Etiology. In my first communication, I based the etiology of fibroma molluscum gravidarum "upon the metabolic changes common in pregnancy." This was but an expression of ignorance. There has been such a great advance in more recent years in the study of the internal secretions and their effects that I find that I must so modify this view as to bring the genesis of the condition into harmony with the scientific notions of to-day.

Properly to understand the relations of the ovary to the other organs of internal secretion, it is necessary briefly to review some facts. Virchow, over sixty years ago, insisted that the ovary, rather than the uterus, is the most important organ of the female organism. This position has been confirmed by all recent studies. Not only has the ovary the function of the production and expulsion of ova, but it has an internal secretion, the evidence of which is seen in the phenomena of puberty and of the menopause and in the effects of complete castration.[3] It is more than likely that the internal secretion is to be found in the corpus luteum which, as Prenant (1) first suggested, takes on the function of a

[2] Am. J. Obst., N. Y., 1906, liii.

[3] One might add that overian therapy in castrated women gives additional weight to the "ovarian hypothesis." Some authors have thrown doubt, however, upon the effect of the ovarian feeding, notably Frank (Arch. Int. Med., 1910, Sept.).

[1] Candidate's thesis for the American Gynecological Society 1913. To which is appended the report of a hitherto undescribed pigmentation of the nails arising during pregnancy.

gland. As Limon (2) states, it is possible that the interstitial gland may account for the internal secretion of the ovary. But so elusive is the subject that while an internal secretion of this organ must be admitted, its exact source is still a subject of speculation. Changes due to this secretion are to be noted in connection with the reproductive processes, such as an increase in the total quantity of blood during pregnancy,[1] and a diminution of the hæmoglobin, for example. As Scheuer (3) points out, changes in the blood evoke metabolic changes which in turn influence the nutrition and nervous state. These, again, are likely to present their primary symptoms in the skin. As instances, the appearance of hair in the axillæ and on the pubes at the time of puberty may be cited, as well as the proliferative changes in the skin of pregnant women.

It is well established clinically, as well as experimentally, that the so-called glands of internal secretion, the thyroid, the hypophysis, the adrenals, and the ovaries, work in correlation with each other. They act vicariously in a way and a hyposecretion of one of them will often in a normal organism give rise to compensatory hypersecretion in another. Clinically, this can occasionally be demonstrated; for example, in a rather gross manner, by the administration of thyroid extract to overcome the amenorrhœa due to obesity.

In continuation of the argument, but limiting it now to the pregnant state, we are confronted by the fact that during pregnancy the ovaries do not functionate aside from the office of the corpus luteum during the first few months. During the remainder of pregnancy the ovaries are in a state of hypofunction. On the other hand, the thyroid (4) and the hypophysis (6) become enlarged while the adrenals (10) take on an increased activity. Lange, for instance, cited by Scheuer (13) made systematic histological examinations of some of the glands of internal secretion during pregnancy and found that there is always a hyperplasia of the thyroid gland; and the hypophysis is similarly affected as demonstrated by Launois (14) and Erdheim (15). Tandler and Grosz (16), as well, studied human subjects who had been castrated with the Röntgen ray and were able to demonstrate an enlargement of the sella turcica. Schenk (17) adds testimony to the same end by his investigations, which show that after castration there is a decided hypertrophy of the cortex of the adrenals. It may be said that while much of hypothesis clings to the subject, we must agree with Hofbauer (18) that we are but at the beginning of things in this domain, and must study with diligence these phases of disease and of altered conditions, clinically and experimentally.

Assuming the truth of these hypothetical considerations and adapting it to the subject in hand, we have an adequate explanation for the appearance of cutaneous disorders during pregnancy. The pigmentation of the linea alba and of the striæ may well be due to the overactivity of the adrenals. Hypertrophic disturbances, such as fibroma molluscum gravidarum, which is, in a way, analogous to the increase in size and in pigmentation in Montgomery's follicles in the breast, may be well accounted for by an increased function of the hypophysis, and the pigmentation of the excrescences by the augmented chromatin thrown into the blood by the adrenals,— the ovarian influence meanwhile being inhibited. This hypothesis is materially strengthened by the fact that with the return of the ovarian function and the restoration to balance of the organs of internal secretion, the growths disappear, while those which remain gradually lose their color. Further, additional testimony is given by the fact that when a woman again becomes pregnant before the fibromata have retrograded, the growths take on renewed activity and even increase in number and become more deeply pigmented.

This hypothesis fits in with the accepted functions of the adrenals. Thus, Cushing (20) advances the view that skeletal overgrowth combined with certain cutaneous changes is an indication of hyperplasia of the anterior lobe of the pituitary body. The anterior lobe does become enlarged during pregnancy, as we have seen, and possibly acts vicariously for the ovary. Cushing's clinical observations and reports substantiate this

[1] Although this has been denied by Fries (Ztschr. f. Geburtsh. u. Gynäk., 1911, xlix, 340).

Fig. 1. Fibroma molluscum gravidarum. A photograph taken eight weeks post-partum.

Fig. 2. Fibroma molluscum gravidarum. A photograph taken eight weeks post-partum.

view in a measure since a stasis or insufficiency of the posterior lobe evokes cutaneous and hirsute changes of the directly opposite type.

Summing up the facts, we can draw the conclusion that *fibroma molluscum gravidarum is one of the hypertrophic disturbances of the skin arising in pregnancy, due to cessation or inhibition of the ovarian function and to an increase in the activity of the pituitary and adrenal glands.* It is a localized process, as is the hypertrichosis (21) occurring in pregnancy, and is dependent upon the same or a similar disturbance of balance of the internal secretions.

The pigmentation of the fibromata is still the subject of doubt and theory, as is, indeed, the subject of pigmentation generally. Solger (22) advances the view that the pigment is due to a biological necessity — the protection of the skin against excessive light during pregnancy. Scheuer (23) points out that we must recognize the relationship between the genitals and the adrenals during pregnancy just as we recognize the relationship between the ovaries and the hypophysis, and the ovaries and the thyroid during pregnancy. And Kroph (24) cites Neu as having found an increased amount of adrenin in the blood during pregnancy and the puerperium (25).

In my paper of six years ago I went thoroughly into a study of cutaneous pigments, but the present state of our knowledge is of no assistance in the solution of the problem except in so far as the adrenals play a rôle, as indicated above.

The Lesions. During the latter half of pregnancy, never before the fifth month, there appear upon the neck and between and underneath the breasts, small sessile, more frequently pedunculated, growths. These vary in size from a pinhead to half a split pea. They are always multiple and are never

grouped according to any specific arrangement. There may be from ten to forty of these growths. Usually they are pigmented, at first of a yellowish color, but becoming yellowish brown or dark brown as the pregnancy advances. Still, some of the growths bear no pigment and one may see every variety of coloring from that of the normal skin to a black-brown. The pedicles are never pigmented, always remaining the color of the normal skin. The smaller fibromata are digitate but the older and larger ones resemble a small mulberry. Although they might be described as polypoid in shape, they bear no resemblance to the ordinary wart, being pointed at the extremities.

Pathology. For the description of the pathological findings and for the microphotograph I am indebted to Dr. F. S. Mandlebaum, pathologist to Mount Sinai Hospital. He has examined several of these growths and from their microscopic study he has written the following description: "The excised tumors are round or oblong in shape, somewhat papillomatous on the surface, and measure from 1.5 mm. to 3 mm. in their long axis. Sections were cut both transversely and longitudinally, and stained by a large variety of special methods, as well as by the ordinary histological stains.

"When viewed by the low power, the sections appear to have a lightly stained central portion entirely surrounded by normal epidermis, except in some places where it is slightly thickened and thrown into folds. In some of the sections which were cut longitudinally a moderate degree of hypertrophy of the prickle-cell layer is noted. The chief feature, however, seems to be a distinct hypertrophy of the papillary layer of the corium. An elongated appearance of the papillæ is noted in places, due to a growth downwards into the corium of the interpapillary processes. With the higher power, the papillary layer shows an increase of the amount of collagenous tissue, the bundles of which are irregularly interlaced, but not closely packed together. In the meshes are numerous connective tissue cells, blood-vessels, and a few lymph spaces. The general appearance of this part of the section is that of a soft fibroma, moderately rich in

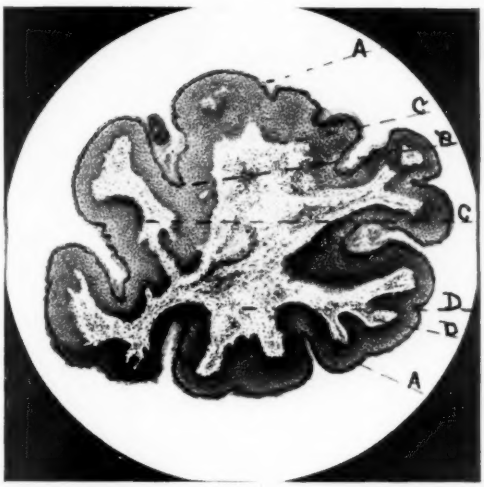

Fig. 3. Photomicrograph of a cross-section of fibroma molluscum gravidarum. A, normal skin; B, hypertrophied papillary layer; C, deposits of pigment; D, young connective tissue.

connective cells. No muscle or nerve fibers can be seen. The base of the tumor at its junction with the normal skin was carefully examined for nerve tissue; but it was also impossible to demonstrate any fibers at this situation. The blood-vessels are rather abundant, most of them being dilated capillaries with thin walls. The connective tissue cells seem rather more numerous in the vicinity of blood vessels than elsewhere, and occasionally a polynuclear leucocyte is seen, but no evidence of inflammation is observed. Here and there a branching connective tissue cell, filled with minute pigment granules, may be seen. Only a few atrophic elastic fibers are present, but no mast cells or plasma cells can be found. Hair follicles as well as glands and adipose tissue are absent from the growth.

"The epithelial elements of the epidermis are all well preserved and quite natural in appearance. The stratum corneum is not apparently thickened, but the layer of prickle-cells in some of the sections shows a slight tendency to proliferation. The cells of this latter in places are rather large, but no mitotic figures are seen. The stratum granulosum is well marked in some places, with abundant keratohyaline granules, while in other situa-

Fig. 4. Pigmentation of the nails arising during pregnancy.

Fig. 5. Pigmentation of the nails arising during pregnancy.

tions this layer can not be distinguished. The basal layer of cylindrical cells is intact, but markedly pigmented. As this layer approaches the normal epidermis at the point of excision, the pigment is no longer seen. None of the pigment granules anywhere in the sections react to the tests for hæmosiderin.

"To classify these tumors properly, a few points must be considered. The chief elements noted are: an absence of hyperkeratosis, a moderate degree of hypertrophy of the prickle-cell layer, an intact basal cell layer, a hypertrophy of the collagenous tissue with atrophy of the elastic fibers, an increase in cellular elements and blood-vessels of the corium, and an absence of nerve fibers. Accordingly, the growth can be placed only under the general type of soft fibromata. The ordinary form of soft fibromata, as well as the neurofibromata, does not come into consideration.

"The verrucous growths (warts) are also excluded on account of the absence of hyperkeratosis. The tumors, therefore, are soft fibromata, and come under the group of fibroma molluscum."

These microscopic findings and the classification were endorsed by Dr. S. Pollitzer, the well-known dermatologist of New York. It will be seen, therefore, that the classification has the sanction of scientific differentiation and is based upon accurate and careful study.

Distribution. The fibromata are distributed mainly about the neck. They are found at the sides of the neck, and in front of it near the sterno-clavicular articulation. They are sometimes seen between the clavicles and

the breasts, or under the breasts; and in one instance they were noted on the back as low down as the angle of the scapula.

In the distribution, too, the condition differs widely from that of the ordinary fibroma molluscum, the distribution of which is general over the body. In fibroma molluscum gravidarum the distribution is exceedingly limited, and is never seen on the mucous membrane, as is occasionally the case with the ordinary fibroma molluscum. I have never seen it about the joints or on the extremities, but DeLee has seen one of the excrescences upon the forehead.[1]

Symptoms and Course. Fibroma molluscum gravidarum differs in no way histologically from the disease when found in males or in non-pregnant women. In its clinical course, however, it departs quite widely from the common type. It usually first appears between the fourth and sixth month of pregnancy as small, slightly pigmented or non-pigmented, sessile or pedunculated excrescences. These increase slowly in number as the pregnancy advances until, at full term, there may be as many as forty or fifty. Or they may appear in limited number, not increasing or diminishing during the further period of gestation.

The previous state of the patient's cutaneous health has no bearing upon the appearance of these fibrous nodules. In one case they developed in a young woman who had formerly suffered from acne and in another they were accompanied by a molluscum contagiosum. But in the other cases which I have observed there was no other accompanying cutaneous lesion nor any history of any previous skin disease.

Within from a few weeks to a few months after delivery most of the excrescences lose their pigment, assuming the color of the adjacent skin, and gradually disappear. The normality or abnormality of the pregnancy plays no rôle in the appearance, development, or disappearance of the fibromata.

No symptoms apart from the eruption are observed. The growths are not sensitive, nor painful, nor uncomfortable, nor do they give rise to itching; nor is there the slightest tendency to any inflammatory process in or about them.

As to the pigment, in some instances it was noted that in brunette women in whom the areola and the linea alba were darkly pigmented, the fibrous nodules, too, were similarly affected. They were of the color of the surrounding skin, however, in many of the cases. In one instance, Mrs. A. J. B., I counted twenty about the neck, of which but six larger ones were darkly pigmented, while the other fourteen were slightly pigmented or were of the color of the surrounding skin. The older the nodules, the more likely they are to be pigmented.

In my first paper on this subject I stated positively that the fibromata disappear with the other regenerative processes of the puerperal state, vanishing within eight or ten months and leaving no scar or trace. In a general way, this holds true. The majority of the excrescences do disappear and in the majority of patients they totally vanish. I have seen in all nineteen patients with this lesion and in fourteen of them no trace of the cutaneous disturbance remained at the end of a year. But in five of them, the lesions did not entirely disappear. Some of the fibromata disappeared spontaneously, many of them lost their pigment, but some remained (in one patient as long as eight years), and some still retained their pigment. But they did not increase in size or number except in the instance to be immediately related.

It is in this respect that I wish to modify my former statement. At present my view would read as follows:

The lesions of fibroma molluscum gravidarum usually disappear as involution from the pregnant state takes place. But in some instances, they remain indefinitely, some of the growths retaining their pigment, some of them losing it.

The case referred to above is, briefly, that of Mrs. A. B., who in her first pregnancy developed a few typical fibromata. Most of these disappeared within eight months postpartum. At this time the patient again became pregnant. The fibromata still remaining took on renewed growth and added pigment and during the course of the second pregnancy increased many times in number.

[1] Surg., Gynec. & Obst., 1908, Feb.

At the present writing — four years after the birth of her second child, the lesions have almost entirely vanished.

Pigmentation of the Nails. As an appendix to this study, I desire to add an observation, which, as far as an exhaustive study of the literature is concerned, is unique. The appended photograph shows the condition perfectly — a longitudinal pigmentation of the nail of the index finger of the right hand and of the middle finger of the left hand. The patient, Mrs. G., is a primigravida. During the later months of her pregnancy she noticed this discoloration of the nails. It was at first of a light brown but became deeper in color as the pregnancy advanced. There was no unusual pigmentation of any of the rest of the body, the linea alba and the areolæ being no more deeply pigmented than usual. Her child is now 15 months of age. The color has gradually disappeared from the nails, but, as the photograph shows (taken in March, 1913), the pigment is still present. It is three sixteenths of an inch in width and is at present of a light yellowish brown color. It will be interesting to note the effect of a subsequent pregnancy upon the nails.

This pigmentation of the nails must be placed in the same group of anomalies of the skin and its appendages as fibroma molluscum gravidarum, without the hypertrophic character of the latter. The appearance during pregnancy, the pigmentation, and its gradual disappearance *post-partum* place it in the same category as disturbances of the organs of internal secretion. As an expression of our ignorance, it is sufficient to say that this manifestation cannot be explained by any facts at present in our possession.

BIBLIOGRAPHY

1. PRENANT. Rev. generale de Science, 1898.
2. LIMON. Étude histologique et histogenique de la glande intertitielle de l'ovaire, Arch. d'Anatomie microscop., Sept., 1902, fasc. ii.
3. SCHEUER. Hautkrankheiten sexuellen Ursprungs bei Frauen, Berlin, 1911, p. 5.
4. WARD, G. G., JR. The Relation of the Thyroid Gland and Thyroidism to the Toxæmia of Pregnancy. Surg., Gynec. & Obst., 1909, Dec.
5. GOODALL AND CONN. The Relation of the Thyroid Gland to the Female Organism. Surg., Gynec. & Obst., 1911, xii, p. 547.
6. FICHERA. Hypertrophie du corps pituitaire consecutive a la castration, Il Policlinico, 1905. Cited by Scheuer.
7. THUMIM. Beziehungen zwischen Hypophysis und Eierstoecker, Berl. klin. Wchnschr., 1909, p. 631.
8. TANDLER, J. Einfluss der Geschlechtsdruesen auf die Geweihbildung der Rennthieren, Anz. der Wiener Akademie, 1910.
9. BIEDL, A. The Internal Secretory Organs (Translation by L. Forster), N. Y., 1913.
10. MARCHAND. Ueber eine eigenthuemliche Erkrankung des Sympathicus der peripheren Nerven, Virchow's Archiv., 1880, lxxxi.
11. STILLING. Ueber die compensatorische Hypertrophie der Nebennieren, Virchow's Archiv., 1889, cxviii.
12. STOERK. Beiträge zur normalen Histologie der Nebennierenrinde, Berl. klin. Wchnschr., 1908, pp. 773 and 908.
13. Loc. cit. p. 100.
14. LAUNOIS. Les cellules siderophiles de l'hypophyse chez la femme enciente, Compt. rend. de la Soc. de Biologie, 1903.
15. ERDHEIM. Ueber einen Hypophysentumor von ungewöhnlichen Sitz, Ziegler's Beiträge, xlvi.
16. TANDLER AND GROSZ. Untersuchungen an Skopzen, Wien. klin. Wchnschr., 1908, p. 277.
17. SCHENK. Ueber die Veraenderungen der Nebenniere nach Kastration, Beitr. z. klin. Chir., 1910, lvii.
18. HOFBAUER. Beitrag zur Aetiologie und Klinik der Graviditaetstoxikosen, Zeitschr. f. Geburtsh. u. Gynäk., lxi, p. 200.
19. Also, Schwangerschafts-toxæmie, Deutsche med. Wchnschr., 1910, No. 36.
20. CUSHING. Concerning the Symptomatic Differentiation Between Disorders of the Two Lobes of the Pituitary Body; With Notes on a Syndrome Accredited to Hyperplasia of the Anterior and Secretory Stasis or Insufficiency of the Posterior Lobe. Am. J. M. Sc., 1913, March.
21. JELLINGHAUS. Voruebergehende Hypertrichosis, durch Schwangerschaft verursacht. Zentralbl. f. Gynäk., 1910, No. 14.
    HALBAN. Ueber ein bisher nicht beachtetes Schwangerschafts symptom (Hypertrichosis Graviditatis). Wiener klin. Wchnschr., 1906, No. 1. Also, Zur Frage der Graviditaetshypertrichose, Wiener klin. Wchnschr., 1908, xxxiii.
22. SOLGER. Die Bedeutung des Pigments für die hellfarbigen Menschenrasse. Dermatolog. Zeitschr., 1905, xii.
23. SCHEUER. Loc. cit.
24. KROPH. Die Erkrankungen des weiblichen Genitales in Beziehung zur inneren Medizin, Vienna u. Leipsic, 1913, p. 448.
25. See also, PICK. Die Ochochronose, Berl. klin. Wchnschr., 1908, p. 591.

THE X–RAY INVESTIGATION OF HABITUAL CONSTIPATION

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SINCE Rieder, in 1904, proved the alimentary toleration of large amounts of bismuth, it has been possible to study the topography and motile function of the intestines in man. The early experiments of Cannon, from 1896 to 1902, upon the gastro-intestinal movements in cats have been duplicated and elaborated in man. The study, by means of the X-ray, of organic and functional disturbances of intestinal motility has received the intimate attention of many men throughout the world.

The X-ray study of constipation demands the use of both fluoroscope and radiograph. The fluoroscope permits the most satisfactory estimation of intestinal motility and mobility. The radiograph simply records the bismuth shadows at the instant of the exposure and it would demand innumerable radiographs taken serially, cinematographically and stereoscopically, to approach the comprehensiveness of direct fluoroscopic inspection. American radiologists have been distinctly slothful in pursuing modern protective fluoroscopy. As a result we find the European literature far ahead of us in the X-ray studies of the gastro-intestinal tract.

The X-ray study of constipation necessitates the inspection of the colon at definite intervals after the ingestion of an opaque meal. Schwarz has recommended the use of a quantity of bismuth with each meal for a period of two or three days.

Both methods demand the faithful record of bismuth shadows by sketch or radiograph and written report, so that the X-ray findings and the clinical data may be correlated and the diagnosis evolved from their collaboration.

A bismuth meal may be compounded in many different ways. The original Rieder meal consists of 40 to 60 grams of bismuth (preferably the oxychloride) with 300 grams of wheat porridge, eaten with a fruit syrup to disguise the bismuth taste. Finding most Americans abhorrent of this bismuth meal, we have turned to the mixing of the bismuth into one pint of fermented milk or a combination of bismuth, acacia, cocoa, milk and water (bismuth oxychloride 2 oz. or barium sulphate (C. P.) 3 to 4 oz., acacia, ½ oz., cocoa ½ to 1 oz., milk 8 oz. Mix these thoroughly and stir into 8 oz. of boiling water and then bring the whole to a boil for a few minutes.) This makes a splendid tasteful suspension of the opaque material and produces good shadows for fluoroscopic inspection.

Frequently it becomes necessary to employ an opaque enema in studying pathological kinks, adhesions, or filling defects of the large bowel. The colon should be prepared for such an examination by thorough cleansing by cathartics and enemas, because fæcal accumulations will interfere with correct interpretations. The opaque enema may be compounded by the thorough mixing of 90 to 150 grams of barium sulphate (C. P.) with 200 grams of Fuller's earth and 1,000 cc. of water. It is best to use a cool enema, as hot enemas often produce an uncontrollable rectal impulse to stool.

The value of the opaque enema depends upon its injection under direct fluoroscopic inspection as the radiograph of an injected colon gives nothing but the map of the large bowel without any data of the filling-defects, presence of adhesions or functional disturbances of spasticity or atony such as fluoroscopy permits. The value of the fluoroscopic opaque enema study can hardly be overestimated if the proper technique is pursued. This demands the use of a protected X-ray couch, called a trochoscope,[1] with the patient lying comfortably upon the back and the protected fluorescent screen upon the abdomen revealing the progression of the opaque enema cæcal-ward. The flexible diaphragm upon the protected X-ray tube box beneath the table permits the inspection of a particular area with a small window, thus approx-

[1] An apparatus for the fluoroscopic examination with the patient in the horizontal position.

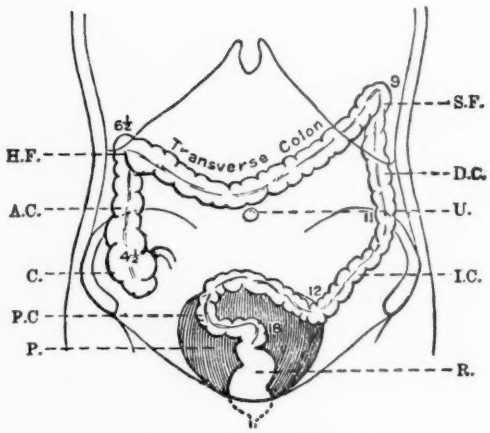

Fig. 1. Diagrammatic sketch of normal large intestine in the horizontal position of the body and with the colon distended by food. The numbers represent the hours after a bismuth meal at which the different parts of the colon are reached. (From Hertz' "Constipation and Allied Intestinal Disorders.") C., cæcum; A. C., ascending colon; H. F., hepatic flexure; S. F., splenic flexure; D. C., descending colon; I. C., iliac colon; P. C., pelvic colon; R., rectum; U., umbilicus; P., pelvis.

imating the application of a higher power lens.

Inasmuch as the value of X-ray studies of constipation depend upon the use of the fluoroscope, let us be mindful of the first principle of successful fluoroscopy, which is, ocular accommodation to darkness. The familiar bellows or funnel type of fluoroscope is absolutely valueless. The open fluorescent screen protected by lead-glass, lead, and aluminum is demanded and this must be used in an absolutely dark room and then only with the eyes accommodated to darkness. The longer one remains in the dark, just so much better do we see the opaque images. It would be a good rule never to switch the current into a tube until the observers had ten minutes' ocular preparation in darkness. All manipulations of apparatus in the dark room should be conducted with red or blue illumination to avoid losing the ocular accommodation to any extent. The importance of ocular accommodation and proper fluoroscopic apparatus bears the same relation to successful fluoroscopy that aseptic preparation bears to successful abdominal surgery.

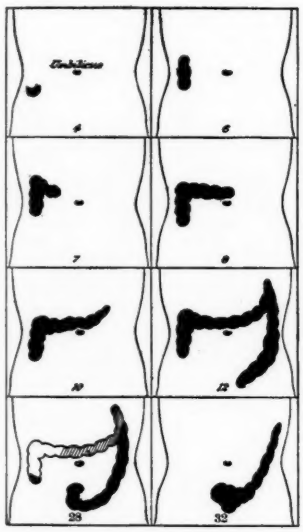

Fig. 2. A series of fluoroscopic colon tracings in a normal individual. The numbers indicate the hours elapsed since the bismuth meal was taken. (From Hertz.) We do not usually find such fillings of the descending colon as depicted above; rather that, as soon as the splenic flexure is passed, the shadows in the descending colon become thin and long with no haustral formation of colon outline and are rapidly passed through the descending colon into the pelvic sigmoid.

The preparation of the patient for an X-ray study of constipation is important. Some radiologists demand that the patient be purged and cleansed by enema preceding the examination. Others, including the writer, prefer that the patient approach the examination in his usual alimentary condition. We know that the colon motility is altered for several days following a thorough intestinal cleansing and thereby may determine faulty X-ray interpretations. Therefore we recommend that intestinal purging and enemas be avoided excepting just previous to an opaque enema. The latter would only be ordered by the radiologist after he had observed the intestinal motility after opaque meals and desired to investigate a filling-defect.

So much for the preparation of apparatus and patient. Now to the X-ray examination of the intestinal motility of habitual constipation. The patient after fasting six hours is first seen to inspect the abdomen and note

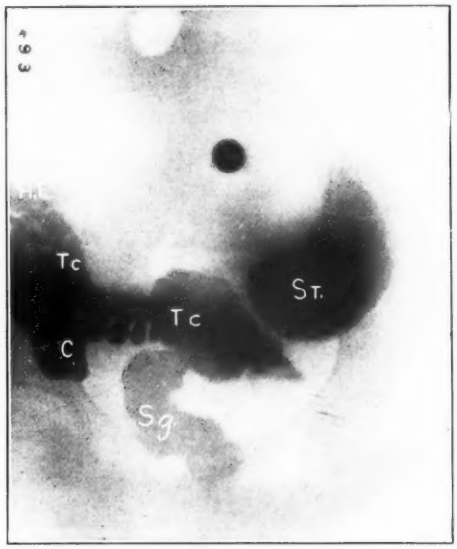

Fig. 3. Bismuth meals forty-eight, twenty-four hours, and immediately before X-ray exposure with the patient standing erect. Note the general visceroptosis (the fluoroscopic inspection of the chest revealed the heart hanging vertically in center of chest). The stomach (St.) presents the greater curvature eight finger-breadths below the umbilicus and the entire stomach shadow to the left of the umbilicus. The proximal colon shows up well filled with the bismuth with the hepatic flexure (H. F.) below the right iliac crest. The cæcum (C.) is low; the transverse colon (T. C.) is very low. There is a fair amount of bismuth shadow in the sigmoid (Sg.). This case is rather typical of the constipation of general atonicity and visceroptosis.

Fig. 4. Bismuth meals twenty-four hours and immediately before this exposure was made with patient erect. Note the dilated, atonic stomach (St.) reaching almost to the symphysis pubis and entirely within the left iliac fossa. The hepatic flexure (H. F.) is four finger-breadths below the right iliac crest and the transverse colon (T. C.) skirts the true pelvis anteriorly. The head of the twenty-four hour meal is at the middle portion of the transverse colon. This is an extreme degree of visceroptosis with atonic constipation.

the presence of air in the colonic flexures, in abnormal loops in the sigmoid and rectum, also to determine the presence of shadows of any previous bismuth medication residues. A bismuth meal is eaten and the stomach is observed as to its filling, peristalsis, size, position, shape, and motility by palpation and voluntary abdominal retraction and pressure. Six hours later the patient is seen to note the emptying-rate of the stomach and the progress of the opaque meal into the colon. The patient should be examined further at the twelve and twenty-four hours interval and upon each succeeding twenty-four hours until the opaque meal has been finally expelled or sufficient data for diagnostic conclusions have been obtained.

The normal rate of different opaque material in the colon differs but slightly. We should remember that barium meals travel a little faster than bismuth meals. A normal colon motility schedule may be constructed as follows: An opaque meal at the end of *six hours* will show the head of the bismuth column reaching to the hepatic flexure where it meets a small gas collection; stomach empty; gas bag (magenblase) of stomach visible beneath the left diaphragm; gas collection in splenic flexure visible; about one-third of the opaque meal in the terminal coils of the ileum and two thirds of the meal filling the cæcum and ascending colon to the hepatic flexure where it appears capped by gas collections. The normal position of these opaque shadows as they fill the respective parts of the intestines, when the X-ray inspection is made in the upright position of the

patient, may be considered as follows: The cæcum lies within the iliac fossa with the lower portion crossed diagonally by the shadow of the true brim of the pelvis; the apex of the hepatic flexure is two to five finger-breadths above the iliac crest; the terminal ileum will be seen rising out of the shadow of the true pelvis and entering the cæcal shadow about four to eight cm. above the lower cæcal border; the remaining terminal coils of the ileum are seen within the shadow of the true pelvis and can only be seen in detail by direct fluoroscopy in the horizontal position; i. e., upon the trochoscope.

Twelve hours after an opaque meal we may consider that normal colonic motility will show the head of the opaque shadows in the iliac colon (designated by older writers as the first portion of the sigmoid); the ileal shadows have entirely vanished into the colon; the hepatic flexure is well filled; the cæcum while still containing opaque masses is not outlined to its fullest extent because there has been a non-opaque meal in the intervening time which has partially entered the cæcum; there may be a gas collection at the splenic flexure, which is only two or three finger-breadths beneath the dome of the left diaphragm, but the transverse colon is completely filled and appears like a string of beads hung from the hepatic flexure at a point two to four finger-breadths above the right iliac crest to the splenic flexure at a point six to ten finger-breadths above the left iliac crest; the transverse colon rarely ascends in a line from the hepatic to the splenic flexure, as our textbook dissections have led us to believe, but usually droops at its center below the umbilicus at least one to two finger-breadths, the degree of drooping depending to some extent upon the amount of opaque material within its lumen; the beaded formation or scalloped outline of the transverse colon is characteristic; the shadows in the descending colon at this twelve hour interval are scant, and appear as disconnected smooth links of thin, small

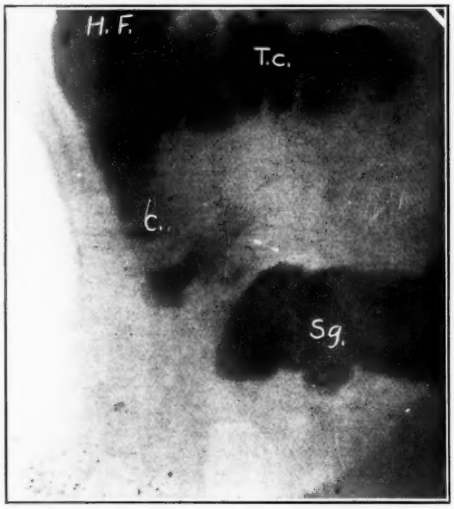

Fig. 6. Twenty-four hours after the last bismuth meal, showing the large sigmoid (Sg.) filling and the well filled hepatic flexure (H. F.) and proximal transverse colon (T. C.). The cæcum shows only faint bismuth shadows, due to the filling of the cæcum with subsequent non-opaque meals. Same case as Figs. 5 and 7.

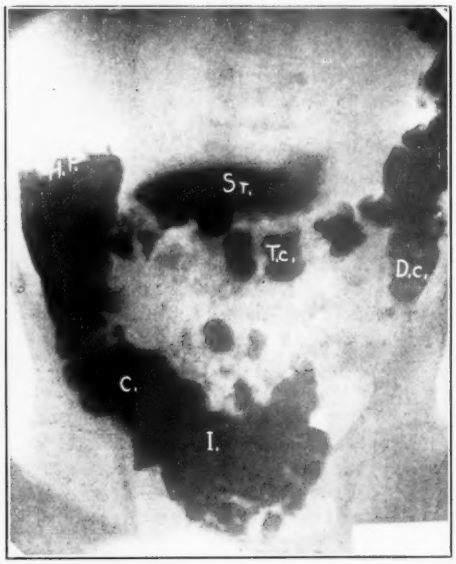

Fig. 5. Bismuth meals twenty-four and six hours before X-ray exposure in upright position of patient. The residue in the stomach (St.) is not due to pyloric obstruction but because the patient ate some food about three hours after the second bismuth meal by mistake. The head of the twenty-four meal has only reached the descending colon (D. C.). Note the shadows in the transverse colon (T. C.) indicative of a spastic condition. Figures 5, 6 and 7 are of the sam case of cæcal constipation.

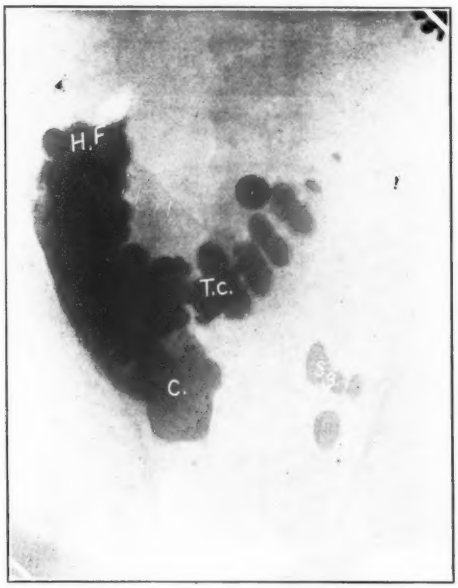

Fig. 7. Bismuth meal forty-eight hours previous. Note the large filling of the cæcum (C.) the hepatic flexure (H. F.) and proximal portion of the transverse colon (T. C.). Small sigmoid (Sg.) residue. This is the same case as Figs. 5 and 6 and the X-ray exposures were made upon successive days. The increased bismuth filling of the proximal colon over that shown twenty-four hours previous is due to antiperistalsis of the colon. These three plates (Figs. 5, 6 and 7) are rather typical of cæcal constipation with a spastic condition of the colon due to the irritation of a mucous colitis.

sausage. At the *twenty-four hour* interval the normal colonic motility reveals the opaque meal scattered along the entire large intestine with a small filling of the rectum; the descending colon shows small opaque elongated shadows but not any large filling, the iliac colon has small sausage-shaped shadows; the pelvic colon (older writers designate this as the second and third portion of the sigmoid) is filled in all its loops; the rectal filling depends more or less upon the time elapsing since the morning meal, as this meal or the drinking of water in the early morning affects considerably the amount of rectal shadow; the cæcum has small shadows upon the circumference of the succeeding intervening meals and the hepatic and splenic flexures reveal gas collections of limited area; the shadows at the hepatic and splenic flexures appear to overlap each other and may be interpreted as kinked if one does not stop to realize that the ascending and descending colon are in a plane much posterior to the transverse colon, which is opposed to the abdominal wall; therefore much latitude must be given to conceptions of kinks at the flexures. We believe this to be extremely important. *Forty-eight hours* after an opaque meal there should be no remaining shadows within the colon with the possible exception of faint opacities in the rectum of fæces about to be expelled.

So much for the normal colonic motility.

Now to the X-ray findings in habitual constipation. First, we must assume or construct some divisions of this subject which we may accept as purely functional disturbances of intestinal motility. We believe that many so-called habitual constipations

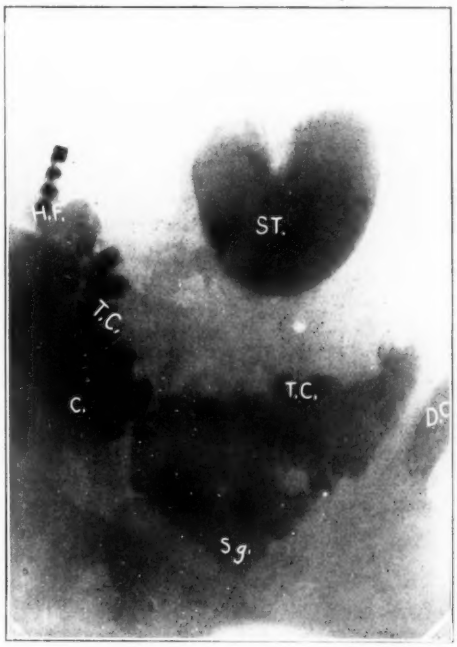

Fig. 8. Dyschezia. Bismuth meal twenty-four hours and immediately before this X-ray exposure in the erect position. The bulk of the twenty-four hour meal has filled the sigmoid (Sg.). See Figs. 9 and 10.

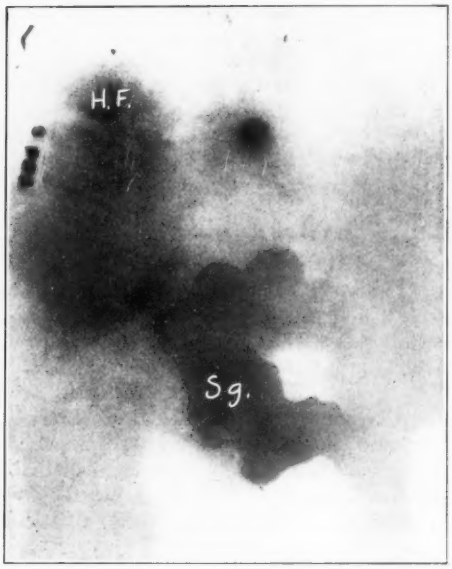

Fig. 9. Dyschezia. Same case as Fig. 8, twenty-four hours later, showing large filling of the sigmoid (Sg.) and scanty shadows in the hepatic flexure. (H. F.)

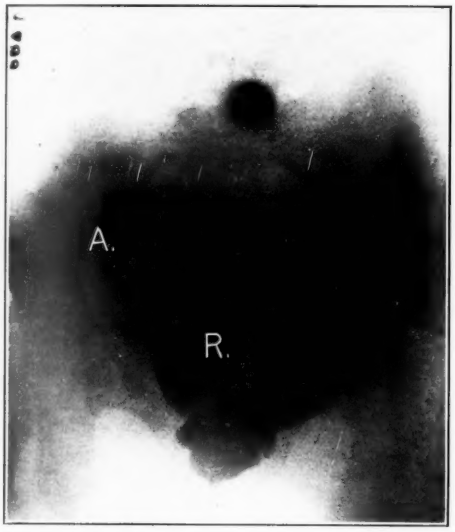

Fig. 10. Dyschezia. Same case as Figs. 8 and 9 Seventy-two hours after last bismuth meal. The rectum (R.) is packed and distended. The appendix (A.) shows a residue.

by clinical estimation are due to some organic kinks, adhesions, loops and mass pathology, but conversely we believe that many cases which are estimated at the present time by our surgical colleagues as stasis constipation due to the above mentioned conditions are only due to atonic conditions of the intestinal and abdominal musculature. We sincerely believe that the careful study of many cases of notable habitual constipation by the opaque meal will reveal this fact, to-wit, that the position of the colon at variance with previous textbook conceptions is not of vital importance; it is the function of the colon in whatever position it may happen to assume that is the crucial test of abnormality.

We are able from our X-ray records to show you case after case where the greater curvature of the stomach reached four to six finger-breadths below the umbilicus and the transverse colon skirted the pelvic brim, where there was a residue in the stomach eight hours after the opaque meal and the colon did not empty in forty-eight hours, and yet these individuals enjoyed their three meals each day and had good daily bowel movements without enemas or cathartics and had no thought of their abdomens. The important point in these cases seemed to be the good tone of the nervous system, the freedom from worry, and a happy, aggressive attitude toward life in general. Now, if these ptotic individuals should lose their "neurotonic" stability, the atonicity immediately developing in the already visceroptotic condition would probably lead some operators to assume that the ptosis and stasis of their nerveless atonic viscera were responsible for autotoxic symptoms, when in reality the restoration of nerve tonus would be all that was necessary for the ptotic individual to reassume normal gastro-intestinal function, although anatomically below par.

Further discussion at this point will only lead us perhaps into the sociological etiology of visceroptosis and habitual constipation and therefore we refrain. But nevertheless we firmly believe that habitual constipation and visceroptosis is as little a medical and surgical topic as it is a topic for sociological, geographical, climatological and neurological

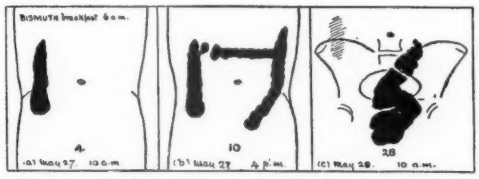

Fig. 11. Typical fluoroscopic sketches in dyschezia made at the four, ten and twenty-eight hour intervals as indicated by the figures upon the sketches. The overloading of the sigmoid and rectum and the good motility of the proximal portion of the colon are the characteristic findings in the X-ray investigation. (From Hertz.)

study. We venture this epigram: that the stomach and intestines can no longer be considered chemical retorts because they are muscular organs whose function depends not upon chemical changes in content but upon muscular progression by neurotonic capacity.

To resume our discussion upon the title of this presentation:

In the first place let us divide our causes of habitual constipation into (1) disturbances of motility by intrinsic or intra-lumenal organic pathology occluding the lumen and thereby the progression of the colonic content such as neoplasms. (2) Disturbances of motility by extrinsic pathology or extra-lumenal adhesions, such as kinks, pericolic membranes, diverticulosis, etc. (3) Disturbances of motility by purely functional disturbances such as hypermotility, hypertonus or from an atypical anatomical position of the colon from faulty mesenteric development or faulty colonic rotation. (We do not believe that the ptosis of the colon succeeds a right nephroptosis, but the converse; i. e., the nephroptosis, dragging pains in the back, right-sided ovarian symptoms and cæcal constipation precede invariably the ptotic and freely movable right kidney and are symptoms dependent upon an overloaded cæcum and hepatic flexure, thereby dragging upon the mesocolon which is so intimately attached to the lower pole of the right kidney.)

In habitual constipation we are concerned only with the functional disturbances of colonic motility and the following subdivisions are suggested: (1) Hypermotility of the colon; (2) chronic obstipation of the ascending-colon type or cæcal constipation; (3) the pure ptosis constipation with low hepatic and splenic flexures and general colonic and bodily asthenia and atonicity; (4) the constipation of the distal colon from lessened rectal reflex, otherwise becoming generally known as the *dyschezia of Hertz*, and (5) hypertonus.

To restate in brief the types of habitual constipation (eliminating similar and dissimilar types due to demonstrable pathological and anatomical conditions), we have those due to:

1. Hypermotility (hyperkinetic).
2. Chronic obstipation of ascending colon type (hypokinetic).
3. Atonic ptosis type (dyskinetic).
4. Dyschezia or rectal constipation.
5. Spastic obstipation (hypertonus).

We feel it necessary to remind you at this point that the pathological processes producing organic obstruction of the colon at various points, such as carcinoma, kinks and congenital malformations, have been excluded from our detailed consideration in an attempt to stick to the original subject.

Now to the X-ray demonstration of these functional types of habitual constipation:

1. Hypermotility of the colon. At the six-hour interval we find, anatomically, a normally placed colon, but the head of the opaque meal has reached the descending and almost the iliac colon; the ileum is empty; gas collections are usually found in the hepatic and splenic flexures. These X-ray findings may be demonstrated also (1) in diarrhœa, in which the subjective manifestations are a differential finding; (2) in gastric achylia, where the subjective clinical symptoms and appearance (cachexia) and an estimation of the acid stomach content are differential points; (3) colonic hypertonus from increased vago-tonic conditions; (4) colonic spasm from mucous colitis and ulceration; and (5) spastic obstipation of unknown (at present) origin (the greedy colon of Hertz). All these demand differential consideration.

With a *hypermotility* displayed at the six-hour interval we note again at the end of twelve and twenty-four hours that the opaque shadows in the cæcal and hepatic areas are now increased in extent and that only a small filling, if any, of the pelvic colon is displayed. The opaque content of the proximal trans-

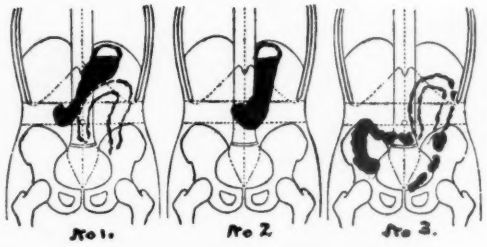

Fig. 12. Fluoroscopic sketches in a case where constipation was due to adhesions. No. 1. Shows an otherwise normal stomach shadow displaced to the right by the gas collection (broken lines) in both limbs of the splenic flexure. No. 2. Shows the normal stomach shadow after the patient had been relieved of the gas collection in the colon. No. 3. Shows the sketch of the bismuth filled colon twenty-four hours after a bismuth meal. The splenic flexure again shows the gas collection, indicated by broken lines. A laparotomy revealed adhesions of the omentum in the right iliac fossa which produced a tension upon the omental apron obliquely across the abdomen to the splenic area.

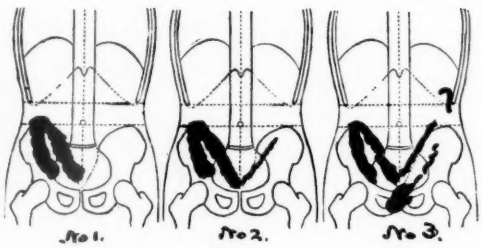

Fig. 13. Fluoroscopic sketches in a case of chronic constipation due to adhesions. No. 1. Shows the blocking of the bismuth meal at the center of the transverse colon and low in the abdominal cavity at the end of twenty-four hours. No. 2. Thirty hours after the bismuth meal we see some of the bismuth meal has passed the site of the constriction but that the shadow is thin, and ascends obliquely to the splenic flexure. No. 3. Forty-eight hours after the bismuth meal there are some bismuth shadows in the sigmoid and descending colon. The site of the kink in the transverse colon was immovable by palpation or by the abdominal contractions of the patient. The operation revealed extensive adhesions between the transverse colon and the sigmoid.

verse colon segment and the descending colon is smaller and massed together into smaller fæcal masses, with increased depth of the haustral shadow; i. e., the usual closely approximated string of large oval beads now appears as smaller round beds with larger intervals between each bead. Topographically and anatomically, the hypertonic colon approximates rather closely our previous textbook conceptions of colon position. Continuing our X-ray investigations of this hypertonic colon we find at the end of twenty-four and forty-eight hours similar conditions as above enumerated, which conditions of increased cæcal and hepatic content are apparently due to the antiperistalsis. This has been demonstrated as a colonic phenomenon by numerous investigators. Sometimes we find at these intervals small strings of bismuth shadows in the descending colon due to the opaque media becoming incorporated with the mucous content of the distal colon, thereby establishing the presence, radiologically, of the mucous colitis so frequently encountered in these hypertonic colons. It is interesting to note how different the X-ray findings become and how the X-ray findings approximate the normal after such cases have persisted with an atropine medication and a frequent bland, fatty diet with regular exhibition of bland oils, paraffine or increased butter fats in the diet.

2. The ascending type of constipation—the hypokinetic cæcal type of habitual constipation. In this type we find at the six-hour interval that the terminal ileal coils are well filled and the opaque meal has only begun to fill the cæcum. There is slow progress at the twelve-hour interval, except that the terminal ileal coils are emptied or nearly so; at least, there is no large ileal residue such as we find with distinct adhesions or peri-ileal bands at the pelvic brim. At the twenty-four hour inspection we find the cæcum filled and distended and probably dropped over the pelvic brim into the true pelvis; the appendix may be demonstrable; the transverse and descending colon are scantily filled with small opaque masses and the general appearance is that of a grossly filled proximal colon and gas filled distal colon. No adhesions of the various colonic parts are encountered, but on the contrary we find the cæcum freely movable in various directions. This is the type of colon in which there is the history of recurring appendicitis in the male and ovarian and tubal symptoms in the female; it is the type of obstipation accompanying the Diettl's crisis of the right nephro-ureteral system. It is inter-

esting to note the change in the intestinal motility of these cases after the exhibition of aloes, strychnine and belladonna, a full diet with recreation, exercise and hygiene, and an increase in the tonicity of the intestinal and abdominal muscles.

3. *The ptosis type* of habitual constipation where only the hepatic flexure is low, and the *congenital ptosis type* where the splenic flexure is low in addition to the right-sided ptosis. These types give practically the same findings in the progress of the opaque media as in the previous type, with the additional anatomical disturbances and low position of the flexures incident to the asthenic, hypotonic, inefficient musculature of the whole body, which is in turn reflected in the gastro-intestinal musculature. There frequently is colonic stasis of five to fourteen days' standing. Yet many of these cases show remarkable changes in the colonic motility under the influence of liquid paraffine or vaseline and rest in bed with small amounts of food at frequent intervals. We have also seen this type submitted to various and sundry operations for fixation of the colon and stomach into textbook configurations without any result other than could be attributed to the good rest and careful diet and hygiene incident to the surgical convalescence. We cannot conceive how the hooking of an atonic tube such as the colon over a figurative nail in the abdominal parietes will benefit its emptying-rate unless you increase the tonicity of the tube by some other extrinsic method and increase the general bodily tonicity.

4. We now come to one of the simon-pure conditions of habitual constipation which has been named by Hertz as dyschezia. Here we have an otherwise normal motility of the colon but a decrease of the rectal reflex resulting in the accumulation of the fæces in large masses within the rectum and pelvic sigmoid. The six and twelve hour X-ray inspection of these cases shows normal colonic motility but the twenty-four hour examination reveals the large bowel with few bismuth shadows excepting in the rectum and iliac sigmoid. This rectal collection of fæcal masses may continue for days; the stool if any is hard and dry and painful. There is an absence of the rectal reflex or impulse to stool which has resulted from faulty hygiene, carelessness, false modesty, rectal fissures, hemorrhoids, etc. This type of constipation is rarely found upon the Continent, where false modesty prevaileth not, but in America and England, where the hygiene of rectal elimination is not a matter of regularity or ease of accomplishment (in Europe, the public lavatory is popular and omnipresent), and the false modesty of over-civilization, are important factors in the development of rectal obstipation or dyschezia. It is interesting to note the X-ray findings in these cases after a rather prolonged course in rectal hygiene following the daily injection of an oil to soften the fæcal masses, followed by enemas, which treatment re-establishes the rectal reflexes. These cases of dyschezia often exhibit a most astonishing array of toxic and neurasthenic symptoms and they are difficult to distinguish from other types previously described without the X-ray study of the case.

We believe that the above four or five types of non-organic, purely functional, habitual constipations are sufficient for the present study. Symptom-complexes closely approximating the above are displayed clinically, but are demonstrated by the X-ray to be dependent upon distinct organic adhesions, kinks, intra-lumenal or extra-lumenal pathology, both malignant and benign.

Furthermore, we maintain that the most satisfactory means of studying habitual constipation is with the X-ray, as by this means alone can we demonstrate the colonic motility and topography to the satisfaction of internist and surgeon. The crucial point is the interpretation of the X-ray shadows which can only be accomplished after study and experience in the normal and abnormal colonic disturbances of motility, function, and anatomy. The X-ray study of colonic efficiency and the contrary is just beginning to reveal to the surgeon and internist a means of diagnosing, prognosing and controlling their therapeutic measures.

In conclusion may I suggest that—

1. The stomach and colon are not chemical retorts but functionating motile organs.

2. The position of the gastro-intestinal tube is not so concerning as its function.

3. The demonstration of actual anatomic alterations of function demand surgery but the surgical treatment of atony is problematical.

4. The subject of the habitual constipation of atonic and congenital origin is a matter of sociological as well as medical investigation.

5. The demonstration of the gastro-intestinal function and pathology by the X-ray is a simple matter compared to the problem of its cure.

BIBLIOGRAPHY

1. Hertz. Constipation and Allied Intestinal Disorders. Oxford Press, 1909.
2. Schwarz. Röntgenoscopy of the Intestines on a Test Bismuth Daily Ration. München. med. Wchnschr., 1912, xxviii, 1313.
3. Schwarz, G. Ueber Hypokinetische und Dyskinetische Formen der Obstipation. München. med. Wchnschr., 1912, lix, 2152.
4. Singer, G., and Holzknecht, G. Ueber Objektive Befunde bei der Spastischen Obstipation. München. med. Wchnschr., 1911, lviii, 2537.
5. Stierlin, E. Ueber die Obstipation von Aszendenstypus. München. med. Wchnschr., 1911, lviii, 1906.

THE VALUE OF THE RÖNTGEN METHOD IN THE STUDY OF CHRONIC APPENDICITIS AND INFLAMMATORY CONDITIONS, BOTH CONGENITAL AND ACQUIRED, ABOUT THE CÆCUM AND TERMINAL ILEUM[1]

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We are presenting here a series of cases which illustrate just how we can make Röntgen diagnoses of surgical conditions in the right lower quadrant of the abdomen and neighboring organs. In practically all of these cases the clinical symptoms were very much alike. First and foremost was constipation, usually extending over a period of years; then there were more or less vaguely localized abdominal pains, with frequent distention and flatulence. In some cases the symptoms pointed more towards the stomach — pain, pressure or distress after eating, heartburn, belching, nausea, frequent headaches. In one case there were vomiting attacks of increasing frequency, until practically nothing could be held on the stomach. Many of these cases had been treated medically for varying lengths of time, both by drugs and by dietetic regulation directed against either the stomach trouble or the chronic constipation. In none of these cases did the treatment give more than temporary relief; in some not even that. In most of them the true condition as shown by the Röntgen examination, and proved by later operation, was not even suspected, although they were under the care of competent internists and surgeons. This was due not to any neglect on the physicians' part, but rather to the inaccessibility of this region to the ordinary methods of diagnosis. We shall not dwell here further on the clinical aspect of these cases, but merely describe their appearances from the Röntgen point of view.

In our examinations we used our usual test meal consisting of 90 grams of bismuth subcarbonate in a mixture of artificially prepared buttermilk and water; in all about 500 cc. In some cases a bismuth enema was used to confirm or clear up the information given by the meal. These cases naturally divide themselves into five groups:

1. *Chronic Appendicitis.* By this term we do not wish to indicate appendices that have necessarily at any time undergone acute inflammation, but refer merely to those cases where the appendix is either kinked or in-

[1] Read before the Surgical Section, Massachusetts State Medical Society, June 10, 1913.

Plate 1. Case 1. This plate shows a large intestine of the typical textbook type. (A) Appendix.

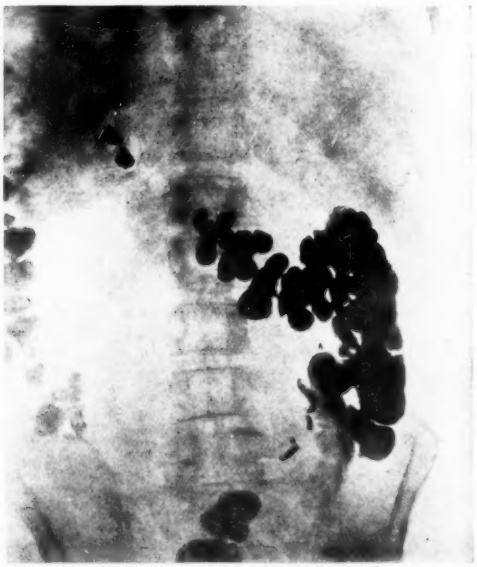

Plate 2. Case 2. Plate made ten hours after the bismuth meal shows ileal stasis with a kinking of the distal portion of the ileum (A). (B) Appendix showing divisions which proved to be concretions.

volved in adhesions. Of course, many of these adhesions are of anomalous developmental origin, but their effect in producing symptoms and their Röntgen appearance are the same as the others. Chronic appendicitis may be shown in two ways; *first*, by the ileal stasis which it produces; and *second*, by actual demonstration of the kinked and adherent appendix. By "ileal stasis" we mean retention of bismuth in the terminal loops of ileum for a longer period than is normal. Normally, six hours after our test meal the bismuth should not only be out of the stomach, but also nearly completely out of the coils of ileum. Many perfectly healthy persons, however, will show a varying amount of bismuth still present in the terminal ileum at the six-hour examination. To be safely classed as real ileal stasis, bismuth should be present at least twenty-four hours or longer. From the presence of marked ileal stasis alone we cannot make a diagnosis of chronic appendicitis; but we can sometimes infer it. It is, however, just this method of inference that has served to bring much of the Röntgen bismuth work into disrepute among careful physicians. We feel that in this, as in other departments of Röntgen diagnosis, the aim should be to find some factor that offers positive, not inferential evidence.

This positive evidence is found in the actual demonstration of the appendix itself; that is, of the bismuth mixture within the lumen of the appendix. The fact that the appendix can be demonstrated Röntgenologically is to-day known to only a small proportion of our surgeons, and even of the Röntgenologists. Only a short time ago Groedel, one of the foremost of German Röntgenologists published a paper in which he claimed to have demonstrated the appendix in three cases, all of which were pathological, and he showed a print of one. From this extensive series he concluded that the normal appendix cannot be demonstrated by the Röntgen method; and conversely, that an appendix which can be demonstrated by this method is pathological. This evidence is of course ridiculously insufficient. Pathological appendices have

Plate 3. Case 2. Plate made twenty-four hours later shows cæcum (A) empty. (B) Appendix still retaining bismuth.

Plate 4. Case 2. Plate made one week later. Bismuth enema, shows (A) patency of ileocæcal valve allowing bismuth to pass back into the ileum. The same relative position of the ileum observed as in plate No. 2. (B) Appendix still full of bismuth from previous examination. Diagnosis: Chronic appendicitis with adhesions about the terminal ileum. Operation confirmed Röntgen diagnosis absolutely.

been shown for many months and prints of them published by Jordan of London who does the Röntgen work for Lane at Guy's Hospital. In this country, Case of Battle Creek over six months ago published a large series of over sixty cases in which he had demonstrated the appendix, some normal, most pathological. Case also inclined to the view that an appendix that can be filled with bismuth, if not definitely pathological, is at least a potential danger. We will agree with him in those cases where bismuth remains in the appendix for forty-eight hours or more, when it has passed out from the surrounding intestinal tract. In the other cases where the size, shape, and position of the bismuth shadow correspond to a normal organ we do not believe that the potentiality for danger is any greater than is always associated with any unremoved appendix. Since our attention was called to the appendix by Case, we have succeeded in demonstrating this organ, either normal or pathological, in about seven out of every ten cases examined. In many cases the normal condition was verified at a later operation for other purposes.

A pathological appendix can of course only show itself, if its lumen is unobstructed, at least in part. It may show a bismuth mass which is sharply kinked in one or more places. There may be adhesions to itself, to the cæcum, ileum, or even to the sigmoid. We have shown an appendix wrapped around and adherent to the terminal ileum so as to obstruct the latter. Retrocæcal appendices can sometimes be shown distinctly. Often these are obscured by the dense mass of cæcal bismuth, and will show up more clearly when some of the bismuth has passed out of the cæcum. In many of the cases bismuth will persist in the lumen of the appendix for hours after it is out of the cæcum and ascending colon: in one of our cases as late as five days after the ingestion of the bismuth meal. Of course, in these cases the appendix is definitely pathological, but usually the abnormal condition is already manifested by accompanying adhesions or kinks.

Plate 5. Case 3. This plate shows a residue in the stomach. Marked ptosis of the transverse colon and ileal stasis (A).

Plate 7. Case 3. This plate shows the effect of adhesions about the cæcum and proximal portion of the transverse colon (A). Appendix (B).

Plate 6. Case 3. Plate made twenty-four hours later showing marked ileal stasis. Ileum (A).

Plate 8. Case 4. Marked ileal stasis after twelve hours, due to Lane kink. Operation. (A) Cæcum. (B) Ileum.

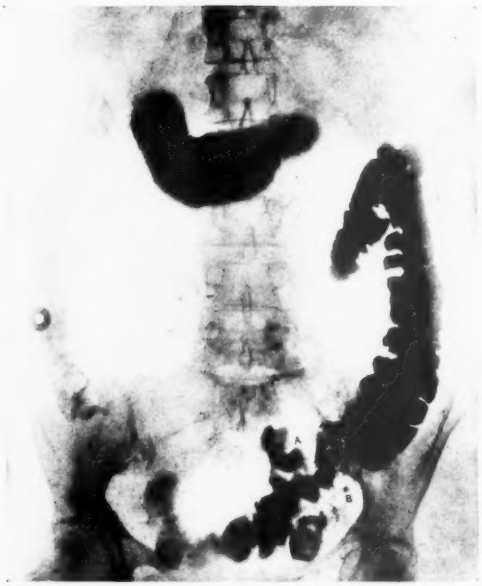

Plate 9. Case 5. There is marked fixity of the terminal ileum due to adhesions. Chronic appendix. Operation. (A) Ileum. (B) Appendix.

Plate 11. Case 6. Plate made twelve hours after the bismuth meal shows marked kinking at the hepatic flexure.

Plate 10. Case 6. Plate made eight hours after bismuth meal.

Plate 12. Case 6. Plate made twenty-four hours later shows marked kinking and fixation between the cæcum and proximal portion of the transverse colon. A indicates the appendix.

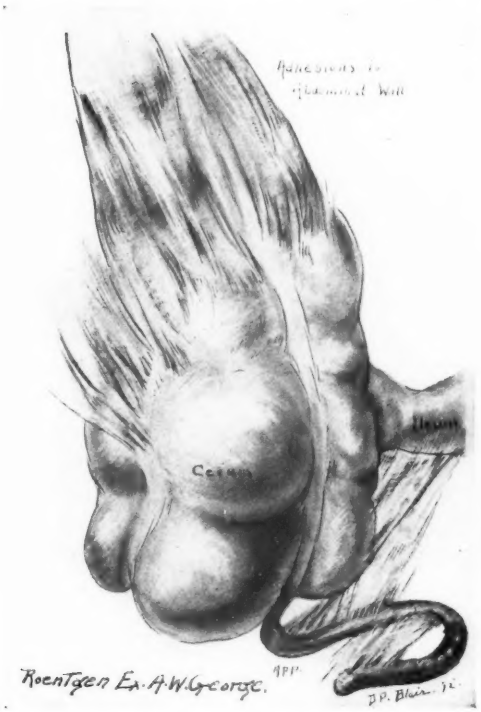

Plate 13. Case 6. Artist's drawing at the time of operation. This case illustrates the mechanism of filling due to adhesions. Röntgen diagnosis: Chronic appendicitis, pericolitic membrane, and general adhesions, confirmed by operation.

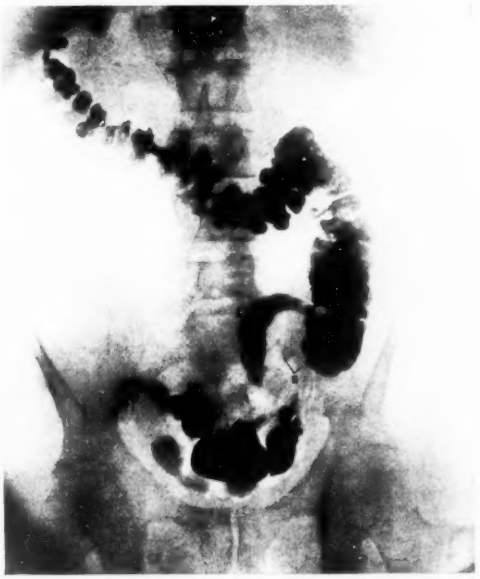

Plate 14. Case 7. Plate showing a fixed terminal ileum. (A) Stasis after twenty-four hours. The distal portion of the appendix is obliterated by disease (B). Operation confirmed the Röntgen findings.

2. *Lane's Kink.* This is the condition so much emphasized by Lane where the terminal loop of ileum is fixed by one or more bands, either congenital, developmental, or inflammatory. This condition, as shown by the Röntgen method, has first of all marked ileal stasis. This alone, however, is of little value. The actual demonstration of the distended and fixed terminal ileum is the real diagnostic point. This loop of intestine will be found in an abnormal and definitely fixed position, at different times during the examination, and even on different days. The fluoroscopic examination here is very important. With the fluoroscent screen we can manipulate and show that the terminal loop of ileum is really fixed.

3. *Jackson's Membrane.* This is sometimes called "membranous pericolitis," and is another condition where opinions are divided as to whether the cause is inflammation or anomalous development. We are not concerned here with the etiology, only with the demonstration of the condition by the Röntgen method. The presence of the membrane which runs across the ascending colon and on to the proximal transverse colon is shown in several ways. First, there will be more or less ileal stasis. This, however, is frequently due to the accompanying Lane's kink or chronic appendicitis. Obstruction in the ascending colon is usually present, and is shown by retention of bismuth in this part and in the cæcum for twenty-four hours or longer. A bismuth enema will also sometimes show obstruction at the same place with perhaps acute angulation of the proximal transverse colon. However, the demonstration of ileal stasis and colonic obstruction is not enough. We must demonstrate the actual effects of the adhesions themselves upon the adjacent parts. Single plates which show apparent "matting together of

Plate 15. Case 8. Plate shows cicatrix of tubercular ulcer at ileocæcal valve (A). Appendix (B).

Plate 17. Case 10. Plate made twenty-four hours after bismuth meal. (A) Kinked appendix. (B) Ileal stasis. Diagnosis: Chronic appendix with adhesions. Operation confirmed Röntgen diagnosis.

Plate 16. Case 9. (A) Showing the effect of adhesions about the hepatic flexure. (B) Chronic appendix, kinked.

Plate 18. Case 11. Röntgen diagnosis: Chronic appendix with obliteration of distal half. (A) Operation.

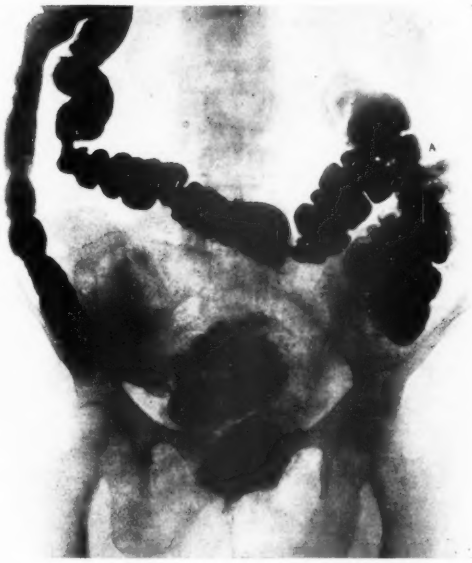

Plate 19. Case 12. Adhesions about the hepatic flexure (A).

Plate 21. Case 14. Chronic appendicitis. Plates show the sigmoid (A) fixed to the cæcum. (B) Ileal stasis.

Plate 20. Case 13. Marked pericolitic veil and adhesions about the hepatic flexure (A). Operation confirmed Röntgen diagnosis.

Plate 22. Case 15. Adhesions about the hepatic flexure and stomach (A). Operation confirmed Röntgen diagnosis.

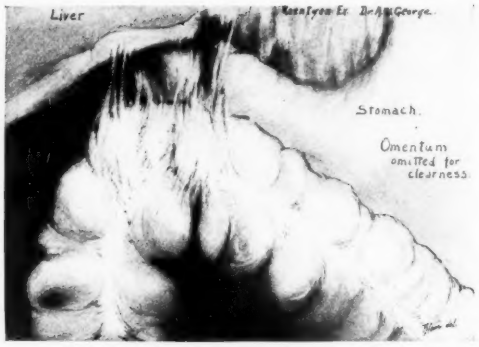

Plate 23. Case 16. Artist's drawing at the time of operation.

cæcum and ascending colon and hepatic flexure" are not conclusive. The diagnosis of Jackson's membrane from this evidence alone is likewise shrewd guesswork. Real positive evidence can only be obtained by manipulation under the fluoroscope where the effect of the adhesions can readily be shown, or by demonstration on plates of the peculiar filling-mechanism which we have found to be associated constantly with Jackson's membrane. When the cæcum is only partly filled with fæcal material the ascending colon and transverse colon may be some distance apart and apparently not connected. However, as the cæcum and ascending colon become filled more and more with ileal contents, these adhesions begin to show their presence by pulling the proximal transverse colon toward the ascending colon. Finally we have the characteristic "double-barrel shotgun" effect of the two adjacent colonic parts. At this time under the fluorescent screen these cannot be separated by manipulation. This pulling together of the ascending and transverse colons as the result of filling is characteristic for these pericolitic adhesions only. With an atonic cæcum (typhlatony) we may have ileal stasis and likewise a dilated cæcum with abnormal bismuth retention. Here, however, we do not have above the mentioned filling mechanism; and we cannot demonstrate adhesions under the fluoroscope. With this condition we have the so-called "ascendens" type of chronic constipation,

Plate 24. Case 17. Cæcum mobile.

which is a subject for medical, not for surgical, treatment.

4. *Adhesions.* This group of cases we will pass over rather rapidly. We have found many cases of chronic constipation where the cause, as shown by the Röntgen ray, was adhesions of the sigmoid to the cæcum from old pelvic inflammation; or the transverse colon may have been adherent to the ascending colon or cæcum as the result of old ulcer, gall-bladder disease, or old appendicitis. In these cases the demonstration of obstruction and ileal stasis was likewise not as valuable as the fluoroscopic demonstration of the adhesions or of their peculiar filling mechanism. In these cases the bismuth enema has been valuable because by its use we have been able to bring the colon more nearly to its normal position under complete filling. Acute angulation due to adhesions show very definitely by means of the enema.

5. *Cæcum Mobile.* This is a condition which, although not found in this particular series of cases, deserves mention here, because of the emphasis which has been placed upon it of late by surgeons like Wilms and

others. This anomaly of colonic fixation can readily be demonstrated by the Röntgen method. The cæcum and ascending colon are found not only to be in an abnormal position, perhaps in the median line, or to the left; but under the fluoroscope they can be very readily moved about. Incidentally, the diagnosis of left-sided appendix can be definitely confirmed.

SUMMARY

In a series of cases which clinically show severe constipation, and more or less indefinite gastric symptoms, examination by the Röntgen ray has shown the causes to be definite surgical conditions in the right lower quadrant. In all of these cases the demonstration of mere ileal stasis or obstruction is not as important as the more positive demonstration of the actual conditions.

In chronic appendicitis the kinked and adherent appendix is shown.

In cases of Lane's kink we can demonstrate the fixed and distended terminal loop of ileum.

In cases of Jackson's membrane we can show the effect of the adhesions under the fluoroscope as well as the presence of a characteristic mechanism of filling.

Various adhesions in this region, as the result of old inflammatory disease or congenital anomaly, can also be shown in the same manner.

Cæcum mobile and left-sided appendix can be readily demonstrated.

PREGNANCY IN THE RUDIMENTARY CORNU OF UTERUS UNICORNIS AND REPORT OF A CASE WITH FULL-TERM FŒTUS[1]

By E. P. QUAIN, M. D., Bismarck, North Dakota

DEFECTIVE union between the two Müllerian ducts in embryo results in various uterine anomalies. A total and permanent separation of the ducts, with the development of a separate uterus tube and ovary from each, is termed a uterus didelphys, or uterus duplex separatus.

The ducts may fuse in the lower segment of the uterus and not in the upper, and a uterus bicornis is the result. Should the fusion be complete for the outer wall of the uterus, but leave a septum between two cavities, we have a uterus septus, or uterus bilocularis.

Again, one Müllerian duct may fail to take any part in the formation of the uterus, while the other duct has formed a complete uterus with a normal tube and ovary. Such an anomaly is termed uterus unicornis. The duct on the defective side does, however, with rare exceptions, form a fairly normal ovary and tube at its upper extremity. The connection between these and the uterus is indicated by a bundle of muscle fibers, often arranged in layers like normal myometrium. Within this muscle tissue lies a small cavity, lined with mucous membrane of endometrial type. The mucous membrane is continuous with that of the tube, but does not communicate with the uterus in more than 20 per cent of the cases observed. The round ligament joins the muscle fibers near the point where the tube is attached.

Into this rudimentary cornu of a uterus unicornis may be deposited a fertilized ovum. In those cases where an open duct does not connect tube and uterus, this impregnation can take place only as a result of transperitoneal migration of the fertilized ovum or of the spermatozoön. The following case from our experience will serve to illustrate these conditions:

Mrs. H., age 21, German, and of excellent family history, came to me for consultation February 28, 1911.

Personal history.—Never ill, menses at 17, regular every 26th day, and painless. Married in December, 1909; last menstruation March 29, 1910, and only slight traces at this time. She had some headache and dizziness during the following two months, but

[1] Read before the Western Surgical Association, Cincinnati, Ohio, December 21, 1912.

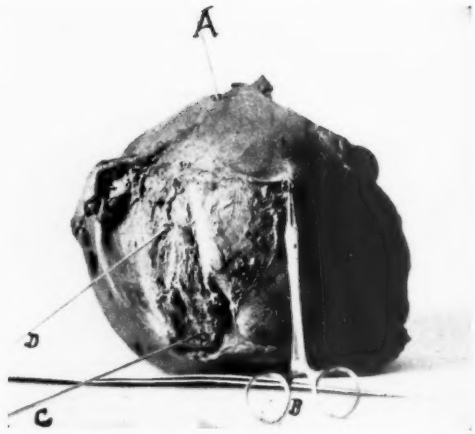

Fig. 1. A, probe at internal ostium of tube. B, forceps on round ligament. C, probe at point of attachment to uterus unicornis. D, probe at point of entrance of main blood vessels.

Fig. 2. Fœtus in cyst with margin of cyst everted. Shows necrotic areas and exposed bones of fœtus, also edges of placenta.

no nausea nor unusual abdominal symptoms. The abdomen enlarged gradually and life was felt in the beginning of August. Fœtal movements were noted daily from this time until December 29th. On this date she had severe intermittent pains in the lower abdomen and expelled from the vagina a "piece of flesh the size of two fingers" and a small amount of blood. After this no fœtal movements were felt. On January 4, 1911, she again suffered from intermittent attacks of pain, and passed a small amount of blood clots. Since then she had suffered considerably from abdominal tenderness, but had been able to do most of her housework. Her abdomen, previously much enlarged, had gradually decreased

Fig. 3. Skiagraph of fœtus showing bone development.

in size, and a hard tumor had developed near the umbilicus. Her breasts, large and distended with milk about January 1st, had decreased in size.

Examination showed a medium size young woman of apparently perfect development; pulse and temperature were normal; heart and lungs normal; breasts large but flabby, and contained milk; abdomen enlarged from a tumor extending from the pelvis to a point slightly above the umbilicus. The tumor was even in outline and had the position, shape and size of a pregnant uterus in the seventh month, but was quite tender and presented a striking woody hardness to the palpating hand. The vaginal mucous membrane was pale, and the cervix hard, closed, and of normal size. A normal size uterus was located below and to the left of the tumor, but attached to it by a thick firm band. The lower pole of the tumor was less tender than the upper parts. Uterine bruit and fœtal heart sounds were absent. The urine showed a slight trace of albumin;

leucocytosis was 10,000. A diagnosis was made of ectopic pregnancy with dead full-term fœtus.

Operation. On opening the abdomen and exposing the tumor, the omentum was found adherent to the anterior wall of the tumor over two small necrotic areas, each the size of a five-cent piece. A dark brown, grumous fluid escaped through these areas after separating the omentum. No other adhesions were present. The right tube was stretched across the upper part of the tumor and the right ovary was attached posteriorly in normal relation to the tube. The tumor was attached to the right side of the pelvic floor by means of a broad, fleshy, and very vascular pedicle. The round ligament entering the tumor laterally about its middle corresponded to the external margin of the pedicle, while the shorter and thicker mesial margin was attached to a normal size uterus about three cm. below the fundus. The round ligament was attached near the inner ostium of the tube.

The left adnexa appeared normal, the ovary being much smaller than the right. From the point of attachment of the pedicle to the right margin of the uterus a membrane extended to the rectum, dividing and partly obliterating Douglas' cul-de-sac.

The tumor, aside from its hardness, had the general appearance of a pregnant uterus, with right tube, ovary, and round ligament situated in a relatively high position, but lacking left adnexa. The uterus, packed into the left compartment of the cul-de-sac, appeared as a normal non-pregnant uterus with normal left tube, ovary, and broad and round ligaments, but minus right adnexa, save the pedicle, 3 cm. long and half as thick, passing from the lower uterine segment upward into the tumor. The pedicle was clamped, and the tumor together with the tube and ovary removed. The blood-vessels were ligated and the peritoneal wound sutured. The abdominal wound was closed, and the patient recovered without complications.

The specimen removed was hard and globular and 22 cm. in diameter. The tube was 15 cm. long, the inner third of which passed obliquely through the tumor wall, while the outer two thirds were free. It was attached across the external surface and near the top of the tumor. The ovary was large and distended from a cyst which was found to be a corpus luteum. The round ligament was attached 5 cm. below the inner ostium of the tube. A triangular raw surface, about 10 cm. to each side, showed the place of excision through the pedicle. The main blood-vessels entered near the outer margin of the pedicle (Fig. 1).

On incising the tumor, a dead, partly macerated and tightly compressed fœtus escaped with some force from the retracting muscular edges of the incision. It occupied a sacroposterior position, with breech presentation. A small amount of thick brownish fluid was contained in the amniotic sac. The thin and necrotic placenta, weighing 300 gm., was partly loosened from the cyst wall and studded with old blood clots. It had a butterfly outline, with the cord 34 cm. in length, attached between the two wings. It was spread over fully two thirds of the cyst wall. It did not reach the top of the cavity, where the necrotic perforations had taken place. These perforations corresponded to the left knee and elbow of the fœtus.

The cyst wall had a thickness of 1½ cm. over its inferior and external surfaces. The superior surface was scarcely ½ cm. in thickness. Its weight, after removal of fœtus and placenta, was 424 gm. Numerous heavier muscle bundles ran in various directions within the wall, leaving thinner spaces between them. No opening or communication existed between this fœtal cyst and the uterus. Microscopic sections made from the pedicle failed to show anything but bundles of muscle fibers, with some round-cell infiltration and a few blood-vessels. The lining of the cyst was too necrotic to show the structure of the mucous membrane. Sections of the wall showed the muscle fibers in the thicker portions to be arranged in strata.

The fœtus, a female, was 45 cm. long and weighed 1915 gm. It was covered with vernix caseosa. Hair, 2 to 3 cm. in length, covered the scalp. The finger-nails extended beyond the finger-tips. One knee, one shoulder, and both elbows were necrotic, the bones protruding through the skin at these points. These parts had been in contact with the contracting muscular cyst wall (Fig. 2).

After these observations we were compelled to modify our diagnosis of ectopic pregnancy and to specify pregnancy in the rudimentary cornu of a uterus unicornis. Additional interest was gained in this instance by the fact that the fœtus had unquestionably lived to term. This is shown not only by the history of the pregnancy, but also by the physical findings on the examination of the fœtus, as stated above. Skiagraphs showing the advanced bone development, and especially the centers of ossification in the lower epiphyses of the femur, add further evidence to this argument (Fig. 3)[1]. That a transperitoneal migration of the spermatozoön, or transmigratio seminis externa, had taken place is established by the finding of the corpus luteum on the same side as the impregnated impervious rudimentary cornu.

A search through the literature disclosed the fact that up to date about 150 cases of this type of ectopic pregnancy have been reported, either directly or referred to indirectly by various authors. Surprisingly few are the reports from American surgeons. It was

[1] See Edgar, Practice of Obstetrics; Olshausen and Veit, Geburtshülfe; Heisler, Text-Book of Embryology.

found practically impossible to classify in a scientific and useful manner the cases reported, and for the reason chiefly that the reports in many instances were quite incomplete. The confusion of terms used in describing congenital uterine anomalies is another obstacle in this connection.

Kehrer (1), in 1900, described 82 cases of pregnancy in the rudimentary horn. Cohn (2), in 1909, added 50 more, but complained that many cases were questionable on account of the confusing nomenclature. Guillaume (3), in 1911, tabulated 145, but does not claim to have exhausted all the literature on the subject. Moreover, a study of his work tends to show that he has undoubtedly included several cases which really were uterus bicornis or uterus septus. It would seem advisable to confine the term uterus unicornis to those cases which have a demonstrable pedicle between the uterus and the cavity inthe rudimentary cornu, where the fœtal sac develops. This pedicle may or may not contain a duct.

Of the reported cases, about 30 are said to have gone to full term (see references 4 to 35). But a closer study of these advanced cases has convinced the writer that this number is much too high. Several authors have described a dead fœtus which had "died at term," while, as a matter of fact, either the history would tend to show that the fœtus died a month or more before full term, or the description of the specimen would fail to prove a development corresponding to full term. Again, the brevity of detail in many of the reported cases prevents us from proving the actual number of full-term pregnancies in the rudimentary cornu on record. This diagnosis must rest very largely on the examination of the fœtus, for it is often impossible to state accurately from the history given by the mother even the approximate time when the fœtus died. This is true especially when symptoms of distress are present both before and after the death of the fœtus. The actual size of the fœtus is of less importance than the development of special tissues and organs. In nearly all the reported cases of unquestionable full-term fœtus the measurements have been below the average for normal pregnancy, while the other signs of full term have been present. This may be accounted for by the excessive pressure within the abnormal fœtal sac and by the deficient blood supply.

When a cavity exists in the rudimentary cornu it has always been found communicating with the tube, and to contain a mucosa surrounded by muscle fibers of a uterine type. Several recorded instances of the formation of hæmatometra in the rudimentary cornu point to such a high development of uterine elements that menstruation is made possible. Under these conditions, the impregnated ovum finds a very congenial soil for development. Fertilization has taken place by transperitoneal migration of the spermatozoön in more than half the cases observed.

The evolution of this variety of ectopic pregnancy is usually quite benign, and it is exceptional that the patient has any symptoms beyond those of normal pregnancy. The rudimentary structure has given no sign of its presence beforehand, and the woman may have had normal pregnancies in the uterus unicornis. The only abnormality noted before the termination of the pregnancy in several cases has been the lateral and somewhat oblique position of the fœtal tumor. The pregnancy continues as a rule until the sac ruptures. The mucous membrane may have defective areas, allowing the chorionic villi to penetrate into the muscle tissue, and to cause intraparietal hæmorrhages and perforation in a manner similar to that of tubal rupture.

If the pregnancy goes to term, labor pains set in. They are less frequent and less severe than in normal labor, and result in nothing more visible than some blood-stained discharge, or perhaps the expulsion of the uterine decidua. The fœtus dies after the first few pains. After the death of the fœtus, the fœtal tumor decreases rapidly in size from the absorption of the fluids. After the cyst wall is firmly contracted about the fœtus and placenta, leucocytic infiltration helps to give the tumor a hardness which can be best described by the adjective "woody." A sudden rupture of the sac may follow the contractions and result in intraperitoneal hæmorrhage and the escape of the fœtus into the abdominal cavity.

The most frequently encountered complication in this condition is a rupture of the gravid sac. It happens most often about the fourth or fifth month. It may happen any time after this period, but very rarely before the third month. A sudden rupture does not usually cause as severe intraperitoneal hæmorrhage as the corresponding rupture of a tube, and for the reason that the muscular cyst walls retract, compress the torn blood-vessels, and grasp firmly the contained placenta after the escape of the fœtus. A fœtus thus expelled from the cyst may live in the abdomen for an indefinite period. It is interesting that the only reported living child delivered from this variety of ectopic pregnancy was removed from the abdominal cavity 19 days after the rupture of the fœtal sac (36). A second case, cited by Cohn, from Reidinger, in my opinion was a double uterus and not a uterus unicornis. Occasionally a more chronic perforation takes place at a point of long continued pressure against some prominent part of the fœtus. The amniotic fluid then escapes into the abdomen and allows the cyst wall to contract firmly upon the retained fœtus. Another less serious complication is the premature death of the ovum within the rudimentary organ. This may be caused by intraparietal hæmorrhage due to penetration of the chorionic villi, or to excessive intracystic pressure.

Retention of the dead fœtus will result in either of the above described complications. If the mother survives the rupture of the sac and the death of the ovum, an absorption of the fluids takes place, and the fœtus, placenta, and membranes become walled off by peritoneal adhesions. The toleration for this foreign body within the abdomen has scarcely a parallel in pathology. Infection and suppuration have proved fatal to the mother in some instances, but more frequently maceration and absorption of the soft parts occur. More unusual results are mummification of the fœtus and transformation into a lithopedion. In several instances necrosis and perforation of adherent organs have caused bones of a retained fœtus to escape into the bowel and into the bladder many years after the time of gestation.

A correct diagnosis of pregnancy in the rudimentary cornu has been made in but a few instances before operation—Draghiesco (37), Hoene (38), Lindner (18), Knauer (39), Wehle (40). The usual diagnosis made is extra-uterine pregnancy. In view of the similarity of treatment, this mistake is not serious. To call this abnormality a uterine fibroid or an ovarian cyst, as has been done a few times, might have dire results. The important diagnostic points to determine are:

1. Whether or not a pregnancy exists.

2. Whether the uterus unicornis can be palpated as distinct from the tumor, but attached to it by a pedicle opposite the internal os.

3. Whether intermittent contractions in the rudimentary cyst wall be palpable as in a normal pregnant uterus.

4. A possible palpation of the round ligament external to the tumor; this would eliminate tubal pregnancy.

The prognosis is better than in other varieties of ectopic pregnancy, owing to the fact that the whole fœtal sac usually can be rapidly and safely removed by clamping and ligating the pedicle. The operations performed within the time of aseptic surgery have been eminently satisfactory.

The treatment is that of ectopic pregnancy in general. The removal of the gravid rudimentary cornu would be indicated when diagnosed, whether the fœtus were dead or alive. If diagnosed in the seventh or eighth month, the question might be raised whether it would be advisable to wait in an attempt to save the child. It is to be remembered that a pregnancy in a rudimentary cornu is less liable to rupture than are the other varieties of ectopic pregnancy. But no temporizing should be practiced unless the patient be put to bed and within instant reach of the operating room. At operation it would seem wrong to exercise a sterilizing technique on the normal tube. Normal pregnancies have followed in uteri unicornes after removal of the rudimentary side, and no instance of tubal pregnancy is recorded.[1]

[1] Mrs. H., the subject of this report, underwent a normal accouchement in April, 1912, thirteen months after the operation. — E. P. Q.

REFERENCES

1. KEHRER. Das Nebenhorn des doppelten Uterus. Dargestellt im Anschlusse an 82 Fälle von Graviditat und 12 Fälle von Hæmatometra. Heidelberg, 1900, p. 159.
2. COHN, M. La Grossesse de l'Utérus Bicorne Unicervical. Rev. de Gynéc. et de Chir. Abdom., 1909, xiii, 3–40.
3. GUILLAUME. De la Grossesse developpée dans une corne utérine rudimentaire. Thèse, Paris, 1911 (Steinheil).
4. BAZTERRICA. Un caso do embarazo en un cuerno uterino con embarazo normal. Semana méd., Buenos Aires, 1903, x, 477.
5. BECKMANN. Weiterer Beitrag zur Gravidität im rudimentären Uterushorn. Ztschr. f. Geburtsh. u. Gynäk., 1911, lxviii, 600–640 (Ref. to Dawydowa-Schepelewa and von Szabo).
6. BLAND-SUTTON. Pregnancy in the rudimentary horn of a unicorn uterus; fœtus of the full term removed sixmonths after its death. Tr. Obst. Soc., Lond., 1902, xliv, 316.
7. CAMERON. Fœtation in a rudimentary horn of a bicornate uterus. J. Obst. & Gynec. Brit. Emp., 1902, i, 67–70.
8. ELBING. Ein Lithopædion im rudimentären Horn eines Uterus bicornis mit Schwangerschaft des anderen Hornes complizirt. St. Petersb. med. Wchnschr., 1890, xv, 299.
9. FRANK. Schwangerschaft im rudimentären Nebenhorn; Ruptur des Sackes. Sitzungsb. d. Gesellsch. f. Geburtsh. u. Gynäk. zu Köln, 1905, Berlin, 1906, 19–23.
10. GALABIN. Fœtation in a rudimentary horn of uterus. Tr. Obst. Soc., Lond., 1896, xxxvii, 225.
11. GALLE. Ueber Nebenhornschwangerschaft. Breslau, 1885.
12. GINSBERG. Schwangerschaft im atretischen Horne eines Uterus bicornis unicollis, eine Variation der Nebenhornschwangerschaft. Breslau, 1891 (P. Cohn).
13. HANNES. Ein Fall ausgetragener ektopischer Schwangerschaft. Monatschr. f. Geburtsh. u. Gynäk., Berlin, 1905, xxi, 356–361.
14. HELLIER. A specimen of uterus with rudimentary horn, from a case where abdominal pregnancy occurred nineteen years before. J. Obst. & Gynec. Brit. Emp., 1904, iv, 441.
15. HOEPFL. Fall von ausgetragener Nebenhornschwangerschaft; Heilung durch Operation. München. med. Wchnsch., 1895, xlii, 756–759.
16. KRULL. Vier Fälle von Schwangerschaft im rudimentären Nebenhorn des Uterus. Arch. f. Gynäk., 1900–1901, lxii, 497–516.
17. LANGLANDS. A case of pregnancy in a rudimentary uterine horn. Intercolon. M. J. Australas., Melbourne, 1903, viii, 129–132.
18. LINDNER. Uterus bicornis mit Gravidität im atretischen Nebenhorn. Zentralbl. f. Gynäk., 1904, xviii, 1408.
19. MACDONALD. Report of a case of pregnancy in the left horn of a bifurcated uterus, with observations upon the nature and the proper treatment of so-called "Missed Labor." Edinb. M. J., 1884–85, xxx, 873–888.
20. MALOM. Ein Fall von Gravidität im rechten rudimentär entwickelten Horne eines Uterus bicornis. Ungar. med. Presse, Budapest, 1897, ii, 495–497.
21. MEYER (LEOPOLD). Zur Pathologie und Therapie der Nebenhornschwangerschaft. Verhandl. d. deutsche Gesellsch. f. Gynäk., 1899, viii, 460.
22. NÜCKEL. Ueber Schwangerschaft im atretischen Horn eines Uterus bicornis unicollis. Monatschr. f. Geburtsh. u. Gynäk., 1899, x, 590–613.
23. OERI. Ein Fall von Uterus pseudodidelphys rudimentarius gravidus. Ztschr. f. Geburtsh. u. Gynäk., 1908, lxxxii, 280–290.
24. REIFFERSCHEID. Ausgetragene Gravidität im rudimentären Nebenhorn eines Uterus bicornis. Deutsche med. Wchnschr., 1907, xxxiii, 1665.
25. ROSTHORN. Nebenhornschwangerschaft: interstitielle Gravidität im isthmischen Tubenabschnitte. Verhandl. d. deutsche Gesellsch. f. Gynäk., 1903, x, 253.
26. SAVAGE. Case of pregnancy in the rudimentary horn of a double uterus; rupture of sac; removal of a ten months' child from the peritoneal cavity. Midland M. J., 1910, ix, 114–116.
27. SCHEFFZECK. Beitrag zur Extrauterine und Nebenhorngravidität, und im rudimentären Nebenhorn. Arch. f. Gynäk., Berlin, 1907, lxxxiii, 422–446.
28. SCHRAMM. Schwangerschaft im linken rudimentären Horn eines Uterus bicornis; Retention einer achtmonatlichen Frucht (Missbildung); Sectio Cæsarea; Entstehung einer linken Ureteruterusfistel; Heilung. Verhandl. d. deutsche Gesellsch. f. Gynäk., 1897, vii, 177–185.
29. TARGETT. Full-term pregnancy in a rudimentary horn of uterus; missed labor; abdominal section and removal of sac; recovery. Tr. Obst. Soc., Lond., 1900, xlii, 276–281.
30. THYWISSEN. Ein Fall von Schwangerschaft im rudimentären Nebenhorn. Bonn, 1900.
31. TREUB. Linksseitige ausgetragene Nebenhornschwangerschaft. Zentralbl. f. Gynäk., 1904, xxviii, 981.
32. WERTH. Retention einer ausgetragenen Frucht in dem unvollkommen entwickelten Horne eines Uterus bicornis. Arch. f. Gynäk., 1881, xvii, 281–97.
33. WILLIAMSON. Specimen of pregnancy in rudimentary horn of a uterus bicornis. Tr. Obst. Soc., Lond., 1902, xliv, 228.
34. WINTER. Gravidität im verschlossenen Horne eines Uterus bicornis. Ztschr. f. Geburtsh. u. Gynäk., xxiv, 160.
35. ZEREZNIKOFF. Sluchai chrevosiecheniya pri beremennosti v zachatochnom rogie dourogie matki zhiznespos obnim plodom. (Abdominal section for pregnancy with living child in the embryonic horn of a uterus bicornis). J. Akush. i jensk. boliez., St. Petersb., 1898, xii, 3–12. Also Monatschr. f. Geburtsh. u. Gynäk., Berlin, 1898, viii, 232–238.
36. V. OTT. Quoted by Zereznikoff.
37. DRAGHIESCO. Quoted by Cohn.
38. HOENE. Quoted by Guillaume.
39. KNAUER. Sitzungsb. Verein d. ärz. Steiermarck. Monatschr. f. Geburtsh. u. Gynäk., xx, 1188.
40. WEHLE. Quoted by Guillaume.

VITAL STATISTICS OF PROSTATECTOMY[1]

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IN a paper read before the Medical Society of the State of New York, in January, 1909, the writer detailed the fatalities occurring in a series of one hundred operations for removal of the prostate gland.

Of these hundred cases, seventy-one were operated by the perineal route and twenty-nine by suprapubic.

Death, as a probable result of operation took place in seven instances, four following suprapubic and three perineal operations.[2]

The statement was made in this paper that "if reliable data could be collected the percentage of deaths directly due to catheter life would probably exceed that of prostatectomy at the hands of competent operators." Even though this statement is accepted as a fact by most surgeons, the medical profession at large has still to be convinced of its truth.

In order to add testimony to the aforementioned statement, I have included in this study the vital statistics of a series of patients suffering from varying degrees of prostatic obstruction who were not subjected to operation, as well as those of the operative cases.

The cases selected for study are only those who have been under my care during the past four years and whose histories I have been able to follow up to the time of death or to the present.

These cases for the sake of comparison have been arranged into two groups: First, those who for various reasons have refused operative relief, and second, those operated upon.

GROUP 1. CASES OF PROSTATIC OBSTRUCTION NOT SUBJECTED TO OPERATION

This group consists of thirty-five patients presenting primary or primary and secondary symptoms of prostatic obstruction of sufficient severity to warrant operation. The division of the symptoms into primary and secondary is an arbitrary one, considering those symptoms produced by the mechanical obstruction; namely, increased frequency of urination, burning, urgency and tenesemus as primary, and anorexia, loss of weight and strength, etc., as secondary.

Five cases of carcinoma are included to record the frequency of its occurrence in relation to benign hypertrophy.

Table 1 gives in brief the salient features.

Summary of Group 1. Thirty patients suffered from benign hypertrophies and five from malignant disease. Seventeen were upon catheter life, using the catheter only during the attacks of acute retention, once or twice a day to relieve overdistention, or were completely dependent upon the catheter. Of these seventeen, fourteen have died, with an average duration of life of two years and ten months after commencing catheter life.

Included in the list is the one exception, namely, a patient who had lived fifteen years after beginning the use of the catheter and died of an intercurrent disease—pneumonia.

The cause of death in twelve cases was directly responsible to suppurative renal lesion, nephritis plus sepsis. One died of apoplexy.

Three are living, two at the end of one year and one at the end of five years.

Of the patients not upon catheter life, the mortality during the same period of observation as with the others, has shown great difference.

Fifty per cent are living and of the fifty per cent who have died, the average length of life after the onset of obstructive symptoms has been four years and eight months.

If any conclusions could be drawn from these cases, it would be summed up about as follows:

Fifty per cent of unoperated patients will die within five years from the onset of obstructive symptoms where catheter life is not neces-

[2] N. Y. St. M. J., 1909, April.

[1] Read before the American Association of Genito-Urinary Surgeons, at Washington, D. C., May 6, 1813.

TABLE I

At Time of Examination			Catheter Life	Residual Urine	From Time of Examination Living—Dead
Name	Age	Duration of Symptoms			
D. M.	69	Primary, 6 months.	No	8 oz.	Living, 1 year.
D. Y.	71	10 years.	No	4 oz.	Died, 1 year, nephritis.
H. T.	72	3 years.	Yes, 1 year	All	Abscess of prostate, sepsis, died.
C. Y.	65	4 years (primary)	No	2 oz.	Died, 18 months, nephritis.
M. R.	65	1 year (carcinoma)	Yes	All	Died, 14 months from onset.
M. C.	49	3 years (fibrous)	No	4 oz.	Died, 4 months from endarteritis.
D. S.	65	3 years (Ac. retention)	During attacks	6 oz.	Died, 12 months from uræmia.
G. N.	74	5 years (Ac. retention)	1 year	All	Died, 1 year from uræmia.
H. T.	58	2 years (primary symptoms)	No	4 oz.	Living 2 years, no secondary symptoms.
L. N.	50	3 months (carcinoma)	No		Died in 3 months.
K. G.	40	6 months (carcinoma)	No		Died in 6 months.
M. E.	35	1 year (carcinoma)	No		Died in 2 months.
E. R.	59	2 years.	No	3 oz.	Living, 4 years.
K. M.	60	1 year.	Yes	8 oz.	Died, 18 months, uræmia.
B. S.	66	2 years (primary)	No	2 oz.	Living and well, 3 years.
H. S.	63	6 months (primary)	No	3 oz.	Living and well, 4 years.
S. N.	67	3 years.	Yes, 2 years	8 oz.	Died, 1 year, sup. nephritis.
C. E.	80	1 year.	Yes, 1 year	All	Died, 3 months, sup. nephritis.
F. G.	71	1 year (carcinoma)	No		Died, 6 months, sup. nephritis.
C. N.	63	3 years (primary)	Yes, in attacks of retention.		Died, following infection from catheter, 2 years.
R. L.	62	6 months.	No	6 oz.	Died, 2 years, nephritis.
R. S.	72	6 months (Ac. retention)	No	2 oz.	Died, endarteritis, 3 years.
M. R.	70	15 years.	Yes, 15 years	All	Died of pneumonia.
M. K.	67	3 years.	Yes, 1 year	8 oz.	Died of apoplexy, 2 years.
H. S.	52	Ac. retention.	Yes, 2 years, off and on		Died, sup. nephritis, 2 years.
B. E.	69	2 years.	No	6 oz.	Living with secondary symptoms.
B. E.	59	2 years.	No	4 oz.	Died, uræmia.
Lee	60	5 years.	Yes, 5 years	8 oz.	Living and well.
K. N.	59	18 months.	Yes, 6 months	20 oz.	Uræmia, 6 months.
L. G.	65	4 years.	Yes, 6 months	4 oz.	Nephritis, died, 1 year.
S. K.	63	1 year.	Yes, 12 months	8 oz.	Living with secondary symptoms.
S. R.	64	6 years.	Yes, 18 months	7 oz.	Died, 2 years, uræmia.
A. T.	61	3 years.	Yes, 12 months	8 oz.	Died, 1 year.
W. N.	73	4 years.	Yes, 12 months	20 oz.	Living with secondary symptoms.
S. V.	68	4 years.	No	3 oz.	Living and well, 2 years.

sary. The beginning of catheter life shortens this expectation of life almost fifty per cent, (2 years and 8 months), and increases the mortality to 66⅔ per cent within the shortened period.

The average length of life of the cases of carcinoma have been thirteen months from the onset of the symptoms.

GROUP 2. CASES OF PROSTATIC OBSTRUCTION SUBJECTED TO OPERATION

This group includes 110 cases, operated upon since 1909, where it has been possible to follow the operative result from the time of operation to the present. Among the living, no case has been included who has not lived at least one year from date of operation.

With the exception of three patients operated by the perineal method, suprapubic intra-urethral enucleation of the prostate has been the operation of election. No two-stage operation has been included in this group.

Ages—

Under 50 years of age	5
From 50 to 60 years of age	26
From 60 to 70 years of age	57
From 70 to 80 years of age	16
From 80 to 90 years of age	6
	110

Thirty-six patients have suffered from primary symptoms only, and seventy-four from primary and secondary. Forty-one had been living catheter lives, exclusive of those who have had the catheter used in the treatment of the bladder and kidneys preparatory to operation.

Pathological changes in the gland —

Symmetrical adenomatous hypertrophy	62 cases
Typical median lobe obstruction	18 cases
Fibrous	17 cases
Carcinoma	10 cases
Tuberculosis	2 cases
Calculous	1 case

Complications. Calculi were present in 14 cases; strictures of the urethra in 3 cases.

Strictures of the urethra complicated the case of calculous prostate.

One case of median lobe obstruction in a Chinaman was complicated by urethral strictures. (Perineal operation.)

One case of symmetrical hypertrophy was complicated by strictures. A primary urethrotomy had been performed four years before the prostatectomy. Recurrent vesical calculi were present before and after urethrotomy. There has been no recurrence of calculi following prostatectomy after a period of four years.

The operative deaths. Exclusive of the ten cases of carcinoma, there have been seven deaths occurring in from twelve hours to one week following operation.

Patient	Age	Time after operation	Cause of death
H. L.	73	12 hours after operation	Shock.
M. O.	68	12 hours after operation	Shock.
M. T.	80	12 hours after operation	Shock, post-operative hæmorrhage.
P. E.	58	5 days after operation	Anuria.
P. R.	71	5 days after operation	Pulmonary embolus.
P. H.	63	5 days after operation	Pulmonary embolus.
B. D.	65	7 days after operation	Anuria.

The patient who died from shock and hæmorrhage had a greatly enlarged prostate removed and subsequent continuous irrigation of the bladder was carried out by means of an indwelling catheter and suprapubic tube. The continuous irrigation so masked the actual amount of hæmorrhage which the patient was having that the patient was *in extremis* before his danger was fully realized.

Up to this time I had used continuous irrigation in occasional cases but this experience made me discard its use. I believe it is safer to allow clots to form and thus check oozing, rather than to take the risk of dislodging them and exciting prolonged bleeding by constant irrigation. Since discontinuing the use of post-operative continuous irrrigation there have been no instances of alarming bleeding.

The two patients who succumbed to embolism had had indwelling catheters inserted shortly after operation, in an effort to expedite wound healing. In both instances, the catheters were not well tolerated and induced very considerable vesical spasm.

I believe that during the straining produced by these spasms, clots were forced out of the pelvic veins into the general circulation with consequent death.

For these reasons it seems that the use of the indwelling catheter after operation should not be instituted until sufficient time has elapsed for the blood clots in the vessels to become well organized.

The above argument also applies to the question of immediate closure of the bladder and suprapubic wounds in an effort to obtain primary union, draining by indwelling catheter only.

I have been able to obtain primary union in three cases by such a procedure, but it would appear that the added post-operative danger produced by an indwelling catheter inserted at an early period scarcely justifies itself.

The two cases who died from anuria presented features in common. They both belonged to that class of prostatiques suffering from overdistended bladders and in whom no infection had taken place. Such a patient in my judgment is the poorest operative risk and requires the more prolonged preparatory

treatment before operation should be undertaken.

Patients in whom a mild degree of cystitis has existed for some time seem to have acquired a certain immunity to the effects of infection and as a rule do better after operation than those in whom the bladder is absolutely sterile.

Patients who have not acquired such immunity before operation and who are in addition suffering from renal disease, are more apt to develop acute exacerbations of a nephritis when post-operative septic absorption takes place.

Two conditions probably are responsible for any pre-existing immunity to the dangers of post-operative absorption, namely, the blocking of the lymphatics by a long continued cystitis and the production of antibodies within the individual. Like observations are familiar in other regions of the body where surgery is undertaken.

Deaths occurring after convalescence from operation to the present time: Exclusive of the cases of carcinoma, there have been six deaths occurring in from two months to three years after operation.

Patient	Age	Time after operation	Cause of death
D. E.	71	Two months	Acute gastro-enteritis.
S. N.	73	Four months	Endarteritis, nephritis.
G. N.	65	Eighteen months	Endarteritis, nephritis.
A. S.	69	Two years	Apoplexy.
W. R.	69	Two years	Endarteritis, nephritis.
H. Y.	63	Three years	Diabetes.

With the exception of the patient who died of acute gastro-enteritis, all of the cases in the above list suffered from advanced kidney disease prior to operation. Each had been upon catheter life before operation and manifested severe secondary symptoms.

OPERATIVE DEATHS, CARCINOMA OF PROSTATE

Patient	Age	Time after operation	Cause
S. D.	66	1 day after operation	Hæmorrhage and shock.
P. T.	65	3 days after operation	Diabetic coma.

DEATHS AFTER CONVALESCENCE FROM OPERATION (CARCINOMA)

Patient	Age	Time	Cause
B. L.	56	4 months after operation	Extensive cachexia.
M. D.	61	7 months after operation	Extensive cachexia.
C. K.	76	10 months after operation	Extensive cachexia.

The patient living ten months had complete urinary retention before operation. After operation to the time of death, he voided without obstruction.

Of the five patients still living, the average time since operation is two years. One patient is alive five years after operation. In all recurrence has taken place, but operation has added so great an amount of comfort to each individual that its performance has been justified.

The patients from whom tubercular prostates were removed are alive at three and two years after operation.

Both are in good general health.

Of the one hundred patients operated for benign hypertrophy, eighty-seven are alive and completely relieved from obstructive symptoms at periods ranging from four years to one year.

Post-operative urinary incontinence is an unknown accident following a properly performed suprapubic enucleation of the prostate.

THE USE OF TUBERCULIN IN THE TREATMENT OF SURGICAL TUBERCULOSIS, WITH CLINICAL REPORTS AND LATE RESULTS[1]

By CARL G. SWENSON, M. D., Chicago
Attending Surgeon Passavant Hospital

A NEW era for the cure of surgical tuberculosis began in 1902, when Dr. Wright of London discovered his opsonic method of tuberculin vaccine therapy.

It is important now after a number of years to review the late results in some of the early cases treated by this method.

This method was founded on the principle of phagocytosis. Blood examinations determined the varied percentages of the phagocytic action of the leucocytes on tuberculosis, and from this investigation the proper dose of vaccine was found for each individual case.

He discovered that the blood plasma contained bacteriotropic substances, one of which was named opsonin because it prepared the bacilli suitably for destruction by the phagocytes, and it is upon the increased amount of opsonin maintained in blood plasma that successful vaccine therapy depends.

The essential requirements for vaccine therapy are proper doses given at the right intervals, and this is accomplished only under the guidance of accurate opsonic tests.

Dr. Koch's failure was due to his excessive doses of tuberculin which caused tissue necrosis and extension of tubercular invasion.

The right dose of vaccine has been found to be from 1/2000 to 1/400 mg. and the best place for injection is on the anterior part of the chest, given on an average every tenth day, and this has been adhered to by the writer for over seven years.

Under too frequent T. R. injections, the phagocytosis is decreased below normal, which is known as the negative phase and the same condition is produced by the administration of too large doses. Both of these forms of treatment, consequently, produce a very injurious effect.

On the other hand, if too small doses of T. R. are injected, the maintenance of increased phagocytosis is not established.

We should determine the proper dose so that no injury be produced and that the greatest possible phagocytic efficiency be obtained.

As a rule, after each proper T. R. injection, the patient in a day or two becomes more lively and cheerful.

Patients with fever should not have T. R. injections for fear of adding fuel to the fire.

The surgical treatment should consist not in an effort to remove all the tissues which may possibly be diseased, but only that portion which is definitely necrotic, since from the report of the cases to follow, as well as from observations of others, one may conclude that healing of extensive tuberculous processes may occur under tuberculin injections without the surgical removal of all the diseased tissues.

It should, of course, be emphasized that the removal of such necrotic tissue is of great importance, and that the vaccine therapy is essentially an auxiliary measure to be used persistently and in combination with such surgical treatment as has been suggested.

One should not become discouraged even if it requires two or three years for a cure, as the result will at last come in the majority of cases.

In cases of mixed infection where slow or no improvement is noticed, autogenous vaccination should be resorted to, or, in other words, vaccine prepared from the bacteria which have caused the secondary infection.

Experienced pathologists in laboratory technique are now found, and anybody who has time and wishes to, can acquire the necessary knowledge in opsonic work, making an accurate administration of vaccine therapy possible.

In the use of T. R. injections, it has been found that an almost safe procedure may be followed without opsonic work by the injection of T. R. 1/1000 to 1/600 mg. every tenth to twelfth day.

But a correct vaccination cannot be made without taking blood tests in the beginning of the treatment and repeating these in one to three months.

Blood specimens of a few drops taken in a U-shaped small glass tube can be sent to a reliable pathologist for examination.

Favorable results with tuberculin injections similar to those which I am about to report have been observed by other workers.

Thus Raw,[2] treated 104 cases of surgical tuberculosis with T. R. with beneficial results.

[2] Lancet, Lond., 1908, Feb. 15th.

[1] Read before the Chicago Surgical Society, April 4, 1913. See discussion p. 514.

Walker[1] after using T. R. in the treatment of a series of cases of genito-urinary tuberculosis, concludes that tuberculin treatment is most promising in primary and secondary vesical tuberculosis, and that it is a valuable adjunct to operative treatment in reno-vesical and genito-urinary tuberculosis.

E. H. Ochsner, in a paper read before the Chicago Medical Society Dec. 9th, 1908, and reported in the Illinois Medical Journal, Feb., 1909, gives most gratifying results with tuberculin in joint tuberculosis. He cites two severe tuberculous knee-joints and a series of cases under treatment, of which the majority are favorably progressing.

His conclusion is as follows: "In tubercular joints uncomplicated with mixed infection, we can, if the patient come to us sufficiently early, secure perfect functional and anatomical results in the great majority of cases."

The use of Marmorek's serum, another anti-tuberculous product, has also met with favorable results in surgical tuberculosis.

Thus Van Huelten from Sonnenburg's Clinic,[2] describes beneficial effects from it, particularly in cases of tuberculosis fistulæ which had resisted other methods of treatment.

Glaessner[3] reports ten cases, chiefly of bone and lymph-gland tuberculosis, which were greatly benefited by the serum. Wühsam, Wein, Hoffa, Lewin, Mannheim and many others, also record favorable results from its use.

IN PULMONARY TUBERCULOSIS

E. Laewenstein[4] gives statistics of 682 cases of all grades of severity treated either with old or new tuberculin or with a combination of both, showing 361 or 52-93/100 per cent in which bacilli disappeared from sputum.

The recent literature on this subject is so extensive that the space of this short paper will not permit a review. I have personally reviewed more than fifty of these articles published during the past five years and many others must have escaped my notice.

By dietetic and hygienic measures, the highest percentage of cures is 20 per cent according to Laewenstein.

To my friend, Prof. Hektoen, I am indebted for much valuable information on Wright's treatment in surgical tuberculosis, and also for the opsonic work and direction given to me by him and his assistants, Drs. Horton, Alice Hamilton, Clark, Davis and Graham.

In order to illustrate the clinical results of this treatment I will give short, concise histories of ten cases treated by this method which I was able to keep under continuous observation and of whose progress a concise history was kept from day to day. One typical history will be given in full in order to show the progress of the disease. The time which has elapsed since this treatment was begun is sufficient to make these observations valuable.

CASE 1. Mrs. C., age 38 years, was admitted to the Passavant Hospital on September 26, 1905.

Previous history: October 15, 1904, patient gave birth to her sixth child. Six weeks later she became seriously ill with severe pains in left inguinal region, hip and pelvis, which continued until July, 1905, when a perforation with pus discharge occurred above Poupart's ligaments.

Patient's condition at time of admission to the hospital was as follows: Legs flexed at about 80° and thighs at 45. Above Poupart's ligament was a swelling 15 cm. in diameter with numerous pus discharging fixtulæ. She weighed about eighty-five pounds.

Vaginal examination revealed a dense resisting mass in left vault.

Her general appearance indicated an approaching death. October 3, 1905, left vaginal vault was incised without any benefit. Following this I opened the inguinal abscess and curetted the whole left iliac fossa, which was invaded from left sacro-iliac joint to ramus pubes with tubercular débris and extensive bone necrosis affecting ramus pubes most extensively. The cavity was disinfected and packed with iodoform gauze.

February 5, 1906, a large abscess had developed in gluteal region. It was opened by an incision 20 cm. long, 3 cm. below external iliac crest; one pint of pus was evacuated and drainage established for iliac fossa.

Believing Mrs. C. to be a hopeless case, I concluded to try Wright's treatment, and on January 20, 1906, gave her the first injection of T. R. 1/1000 mg. These injections were given every two weeks at first and later on about every tenth day, and the dose was increased to 1/600 mg.

The opsonic work was done twice every week for a year by Prof. Hektoen's assistants, Drs. Horton, Hamilton, and Clark.

After two months' treatment with T. R. the patient became more buoyant. Her daily improvement was noticed and her recovery astonished all who came in contact with her, and by some it was thought phenomenal.

In order to correct the deformity, extension by weights and pulley was first applied to the right leg, which finally became straight.

The same treatment was applied intermittently to the left leg, but with less success. The patient was unable to endure extension continually because the left hip joint was firmly ankylosed.

In August, 1906, the patient commenced to sit up for one to two hours daily, and Sept. 1st, 1906, she commenced to walk with crutches. Her average opsonic index was 2, rising at times to 3.

January 30, 1907, after seventeen and one-half months in Passavant Hospital the patient was discharged as cured. Her left leg was 10 cm. shorter than the right, due to the ankylosis of hip joint. The wound was almost healed;

[1] Practitioner, Lond., 1908, lxxx, 723.

[2] Deutsche Ztschr. f. Chir., 1906, lxxxiv.

[3] "Über das Marmorek serum," Deutsche med. Wchnschr., 1908, xxxiv, 1261.

[4] Über Tuberculinpraparate zu diagnostischen und Heilzwecken. Handbuch, Immunitätsforschung, erganzungsband, 1911, p. 350.

her weight had increased to one hundred and thirty-seven pounds, a gain of fifty-two pounds.

In May, 1908, the patient returned to Passavant Hospital and gave birth to her seventh child. In March, 1910, she gave birth to her eighth child.

The patient continues in perfect health and does her housework for eight children.

CASE 2. Mrs. B., age 40, came under treatment May 18, 1906. Previous history: In June, 1903, she developed tubercular peritonitis. For this she came under my care at Passavant Hospital February 1, 1904. A laparotomy was then made, half a gallon of serous fluid, both Fallopian tubes, right ovary, and the appendix were removed. Her whole abdominal cavity was invaded with miliary tubercular nodules and there were severe intestinal adhesions. She left the hospital April 12th, with an abdominal fistula for which she returned to the hospital June 7, 1904. The 26th of this month there appeared in the wound several fæcal intestinal perforations, and she had from this time on no rectal evacuations until following September 9th, when a second operation was performed. The incision was made in left lumbar region external to the rectus muscle and an ilio-colostomy at the junction of transverse and descending colon was performed. The result from this operation was only partial relief. About one third of the amount of fæces continued to pass through the wound during the following eight months. Then at the third and final operation, two intestinal fistulæ were found and they were closed with purse-string sutures. A loop of intestines six inches in length adherent to the wound was also excised. After this last operation patient had normal rectal fæcal passages.

After having remained 22 months in the hospital she returned to her home with an unhealed abdominal fistula. For this condition the tuberculin injections of 0.001 to 0.002 mg. were given every tenth to fourteenth day for a year. After six months' time of tuberculin injections the fistula was closed, and it has remained cured now seven years.

CASE 3. Mr. S. K., age 27, commenced treatment February 26, 1906. Previous history: At five years of age patient was treated for tuberculosis of hip by Dr. Fenger. Two years later he was operated on and the femoral head removed. The wound never healed. At the age of 14, in May, 1899, when I began to treat him, he had a large abscess of right hip and thigh incised. Wound healed except three fistulæ which remained unhealed. One of these fistulæ extended by its sinus from the right anterosuperior iliac crest through the iliac fossa into the posterior inferior part of the bladder.

During May and June, 1903, he had otitic meningitis from which his hearing was lost.

After the first two tuberculin injections he became more buoyant and he said: "The leg never has felt so good as it does now." In two months' treatment with tuberculin injections, one sinus was healed and in the ten months the second sinus had healed. But the third sinus extending into the bladder seemed unyielding to all resources.

Prof. Hektoen had him under his personal care for nine months, from January to October, 1907, and during three months of the summer that year patient had not a single tuberculin injection, nevertheless his opsonic index from blood tests made every week always averaged 2.

October 28, 1907, I began the tuberculin treatment which was continued interruptedly until February 4, 1910. All kinds of local treatment to the third sinus extending into the bladder were used without benefit. Biers' treatment was tried and also Beck's paste. The paste proved very disastrous and according to my experience should never be injected into urinary organs. As a last resort I injected a 10 per cent solution of argyrol into the sinus a few times and after its use the sinus finally healed. After four years' treatment the patient remains now cured over three years.

TUBERCULAR CERVICAL GLANDS

CASE 4. Miss A. J., age 24, commenced treatment May 13, 1907. At 12 years of age patient developed tubercular glands on the right side of neck. For these diseased glands she had four operations of which the last one had been performed by a very eminent surgeon, in a thorough, extensive and radical manner. Eight months later she had facial erysipelas with subsequent recurrent enlargement of the cervical glands and six months after this attack she came to me for treatment.

At this time her right cervical glands below the inferior maxilla were as large as a fist, and in front of the ear was one gland the size of an egg. After 17 months' treatment with tuberculin patient has now remained cured five years.

CASE 5. Master E. E., age 7, developed tubercular glands on the left side of neck. These glands enlarged to the size of hazelnuts and they formed an abscess which was incised. After 18 months, having had 43 tuberculin injections, patient has now remained cured six years.

CASE 6. Mr. M. A., age 30, commenced treatment February 10, 1908. At this time he had a large tubercular inguinal abscess, which extended upwards to the umbilicus. The abscess was incised in Passavant Hospital and treated in usual manner. After a year's treatment with tuberculin he has now remained cured four years.

CASE 7. Master E. L., age 17, commenced treatment March 12, 1908. At this time the boy had large tubercular glands on both sides of his neck. After four months' treatment with tuberculin, all the enlarged glands had disappeared. Number of tuberculin injections given were 35. Patient has now remained cured five years.

CASE 8. Master C. S., age 7, father died of pulmonary tuberculosis. Commenced treatment in June, 1909. The boy at this time had very large tubercular cervical glands and the conjunctivæ and cornæ of both eyes were ulcerated and severely inflamed. After two months' treatment with tuberculin the ulceration of both eyes had disappeared. The tuberculin injections were continued, in all, 15 months, and the dosage given varied from mg. 0.0005 to 0.001. He is now a vigorous, strong boy and remains cured three years.

CASE 9. Mr. A. B., age 30, developed when twenty-one, tuberculosis of the right cervical and axillary glands. For these diseased degenerated glands he had during the following two years four operations. Then he was referred to me for tuberculin injections and these were given him for a period of 21 months. The number of injections given was 63. During my treatment, patient had three operations for extensive tubercular invasion from right axillary glands along the whole posterior fascia of the pectoral muscles to the clavicle. This patient has now remained well for four years since the conclusion of this treatment.

CASE 10. Miss C. G., age 40, commenced treatment July 19, 1909. Patient at this time had very extensive tubercular degeneration of the cervical glands on both sides of the neck from the mastoid processes downwards to below the clavicles. There were numerous sinuses which necessitated continuous local treatment during the whole time that the Wright's vaccine therapy was given to her. Time of tuberlin treatment was two years and number of injections given was 42. Patient has now remained cured two years.

I have personally examined cases 1, 2, 3, 4, 5, 8 and 10 in this series within a month, and have

reliable information concerning cases 6, 7 and 9, who reside at a distance.

As a result of my experience in these and many more recent cases of surgical tuberculosis treated with tuberculin, I would propose the following conclusions:

1. In my experience, tubercular gland infections that have not entered the stage of caseous degeneration have yielded to tuberculin treatment.

2. Almost all local tuberculosis may be cured under persistent, skillful, surgical treatment, assisted by tuberculin injections properly given.

3. Tuberculin injections may be safely given every tenth or twelfth day without opsonic work; of course it is preferred that one be guided by occasional blood tests.

4. All tuberculin injections should be given by a reliable, clean person, and the best place for the injections is on the anterior part of the chest.

5. Duration of tuberculin treatment; one half to two years.

6. All hygienic treatment, such as fresh air, good food, and attention to digestive organs, is of vital importance.

7. For tubercular sinuses extending into the urinary bladder the bismuth paste injections are usually contra-indicated.

The following articles contain much important material, but the limit of this paper makes it impossible to give a fair review.

1. K. Dluski. Einige Bemerkungen über die specifische Therapie der Tuberkulose. Beitr. z. Klin. d. Tuberkulose, xvi, 71–182. Anwendung des Beraneckschen Tuberculins. T. R. in der Tuberkulose.

2. Junius. Tuberculin Treatment of the Eye. Ophthalmology, 1910, January.

3. Watson, C. G. Treatment of Tuberculosis of the Knees. Practitioner, Lond, 1910, March.

4. McArthur, L. L. Vaccine Therapy in Surgical Tuberculosis. N. Y. St. J. M., 1910, May.

5. MacLeod, T. and MacLeod, N. K. Treatment of Surgical Tuberculosis by Vaccines. N. Y. St. J. M., 1912, xii, 1–54.

6. Kraemer, C. Tuberculin in After-Treatment of Surgical Tuberculosis. Med. Klin., Berl., 1908, x, 111–142.

7. Stark, J. Surgical Tuberculosis and Opsonic Index. Brit. M. J., Lond., 1907, June 29.

8. Wilms, M. Tuberculin Treatment in Surgical Tuberculosis. Deutsche med. Wchnschr., 1911, xxxvi, 1633–1680.

9. Gaines, L. M. Vaccine Therapy in So-called Surgical Tuberculosis. Atlanta J. Rec. Med., 1911, March.

10. Rosenbach, F. J. Erfahrungen über die Anwendung des Tuberkulin Rosenbach bei chirurgischen Tuberkulosen. Deutsche med. Wchnschr., 1912, xxxviii, 585–632.

11. Quervain, F. de. Les principes modernes dans le traitement des tuberculoses dites chirurgicales. Semaine méd., Par., 1911, xxxii, 433–444.

THE HISTOGENESIS OF CANCER (CARCINOMA) OF THE BREAST AND ITS CLINICAL SIGNIFICANCE[1]

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THE author desires that the subject of histogenesis be not confused with etiology. No direct reference to etiology will be made, although conditions which are closely associated with histogenesis will be described in order to give clinicians some knowledge upon which to base empirical therapeutics in view of no definite or absolute knowledge of any specific treatment for mammary carcinoma.

The subject as dealt with in this study has double significance. It is presented as additional knowledge to the general pathology of carcinoma, i. e., to the apparent evolution of perverted, migratory and destructive epithelial cells (carcinoma) from cells which, under normal conditions, run a definite and rigid life-course in a cellular communistic life of well-being.

The second feature will assist teachers and clinicians in understanding more clearly the logical sequence of at least a part if not all of the parenchyma of the epithelial neoplasms, which plays such a large rôle in the science of medicine.

This investigation was stimulated by the lack of applicability of published descriptions of histogenesis and the existing terminology of carcinoma of the mammary gland to the vast material, which has demanded clinical significance from the writer.

Clinicians demand from the scientific world facts and utilitarian deductions. Their main object is the eradication or amelioration of physical suffering and their patience is often taxed to the extreme by the overwhelming onslaughts of neoplasms, which only the courageous have seen fit to constantly, empirically treat, first this way and then that, in hope of ultimate victory. The progress of the surgeon's efforts has been greatly impeded by a rich but chaotic terminology for which he is partly responsible on account of insufficient knowledge of normal and pathologic processes and conditions. The pathologist, however, has shared in the production of this chaos by his withdrawal from the immediate field of the clinical activities of his practical colleague.

The clinician deals with specific and individual needs while the general pathologist has by or on account of his withdrawal found his problems general or purely biologic.

There are twelve apparent synonyms for the condition which Koenig (1) described as "mastitis chronica cystica." They are found in American, English, German, French and Italian literature and are doubtless to be found in the literature of other countries which have derived scientific inspiration and information from those already named. Unfortunately for the readers, especially the practitioner, all twelve synonyms do not appear in the same article. One writer utilizes one term and another utilizes another. Discussion, strife, misunderstanding and discouragement have followed acceptance of various terminologies without a complete knowledge of the complete list of synonyms.

This extravagance of synonyms is but one example of the chaotic condition of terminology of pathologic mammary conditions. It is unharmonious, almost always undescriptive, semi-clinical, semi-scientific, often local in application, and frequently requires some knowledge of the life and works of some investigator for explanation.

Terms which embrace the names of abnormal processes, the physiologic processes of which are extremely indefinite, are utilized—"abnormal involution" (Warren, 2), and "senile parenchymatous hypertrophy" (Bloodgood, 3).

In the presence of such an indefinite condition of our knowledge one finds textbooks and journals which continue to publish classifications of mammary tumors, especially carcinomata, which are dissimilar and are

[1]An abstract of this paper was read before the Southern Surgical and Gynecological Association, December, 1910.

based on so-called definite entities. Thus many practitioners believe that "scirrhous carcinoma" and "medullary carcinoma" are two different things and indeed attribute very definite prognostic differences to each. When one adds to these terms the so-called "*comed carcinoma*," "*adeno carcinoma*," "*encephaloid carcinoma*," "*carcinoma simplex*," "*cyst-adeno-carcinoma*," "*carcinoma gelatinosum*," and "*carcinoma sarcomatodes*" the practitioner, who usually is not and cannot be conversant with all medical and scientific literature, necessarily either gives up trying to classify and name his own material or loses faith in the present presentation of the subject of pathology by pathologists. The lack of faith is well earned by the pathologist, who allows his profession to be maligned by the present utilization of internes, as pathologists, who innocently carry out the work of their partially trained teachers, who themselves have written or taught the above mentioned and criticized chaotic terminology and classifications. As proof of the existence of terminological extravagance one needs only to examine the following list, which is a compilation of the terms utilized in such excellent works as Adami and Nicholls (4) "Special Pathology," Kaufmann's (5) "Spezielle Pathologische Anatomie," 1911, Aschoff's (6) "Spezieller Teil, Bd. II, Pathologische Anatomie," Borst's (7) "Die Lehre von den Geschwülsten," Ziegler's (8) "Lehrbuch der Speziellen Pathologischen Anatomie," E. Forgue's (9) "Precis de Pathologie Extreme" (Collection Testut), The American Textbook of Pathology (10), and the textbook by Delafield and Prudden (11).

Mastitis: diffuse or interlobular
Acute galactophoritis
Chronic mastitis
Cirrhosis mammæ
Mastitis carcinomatosa
Fibroma
Adenoma
Fibro-adenoma
Adeno-fibroma
Cyst-adenoma
Lipoma
Myxoma
Myoma
Angioma
Osteoma
Chondroma
Epithelioma
Carcinoma
Sarcoma
Adeno-sarcoma
Adeno-myxoma
Pericanalicular fibroma
Intracanalicular fibroma
Intracystic papilloma
Fibroma cysticum
Adenoma acinosum
Adenoma tubulare
Scirrhus carcinoma
Medullary or encephaloid carcinoma
Adeno-carcinoma
Cystadeno-carcinoma
Simple parenchymatous mastitis
Mastitis infectiosa parenchymatosa
Mastitis infectiosa interstitialis
Mastitis chronica cystica
Maladie cystique de la mamelle
Cystadenoma polycystoma
Intracanalicular cystadenoma
Mastitis tuberculosa obliterans
Pure adenoma
Fibro-adenoma acinosum
Fibro-adenoma tubulare
Fibro pericanaliculare or plexiform fibroma or fibro-adenoma
Pericanalicular fibro-myxoma
Fibro-myxosarcoma
Fibro-adenoma cysticum
Fibroma intracanaliculare mammæ œdematosum
Retention cysts
Solitary cysts
Galactocele
Dermoid cysts
Epidermoidal cysts
Cysto-sarcoma or cysto-sarcoma phyllodes
Fibroma myxoma or sarcoma proliferum or arborescens
Sarcoma papillare or polyposum
Cystadenoma papilliferum (i.e., intracanalicular cystadenoma)
Cystic papillary epithelioma (fibro-epithelioma)
Fibromyxoma
Myofibroma
Hæmangioma
Enchondroma
Osteochondroma
Round cell sarcoma
Round and spindle cell sarcoma
Pure spindle cell sarcoma
Polymorphic cell sarcoma
Giant cell sarcoma
Angiosarcoma
Melanosarcoma
Alveolar sarcoma
Chondrosarcoma
Endothelioma
Osteoid sarcoma
Sarcocarcinoma
Chondro-sarco-carcinoma
Carcinoma chondrosarcomatosum
Carcinoma solidum
Carcinoma solidum simplex
Carcinoma solidum medullare
Carcinoma solidum scirrhosus
Carcinoma colloides
Carcinoma cysticum
Carcinoma cylindromatosum
Adeno-carcinoma cylindromatosum
Cystocarcinoma papillare

Cystocarcinoma simplex
Cystocarcinoma papilliferum
Carcinosarcoma
Carcino-chondrosarcoma
Cancer en cuirasse
Acinal carcinoma
Fibro-adenoma pericanaliculare
Fibro-adenoma intracanaliculare
Adeno-myxofibroma
Adeno-lipo-fibroma
Adeno-fibro-sarcoma
Cystadenosarcoma phyllodes
Solid adenoma
Cystadenoma papilliferum
Blood cysts
Milk cysts
Butter cysts
Involution cysts
Psammocarcinoma
Colloid cancer
Carcino-sarcoma
Melanoma of the nipple
Perithelioma
Hypertrophia vera mammæ
Mamma lactans
Fibro-adenoma mammæ.
Adenoma mammæ
Fibro-adenoma mammæ peri- et intracanaliculare
Tubular adenoma
Cystadenoma (phyllodes) mammæ
Cystadenoma (mammæ) pseudo-papilliferum
Adenoma (mammæ) proliferans
Carcinoma scirrhosum
Carcinoma gelatinosum
Carcinoma adenomatosum
Carcinoma medullare
Fibroma pericanaliculare
Fibroma intracanaliculare
Adenoma mammæ acinosum
Adenocystoma
Adenocystoma papilliferum
Fibroma (sarcoma) phyllodes
Adeno-myxofibroma
Adenosarcoma
Cystoma mammæ papilliferum
Intracanaliculare papillary fibroma or papillary adenocystoma
Intracanalicular fibrosarcoma
Cystocarcinoma mammæ papilliferum
Papillary cystocarcinoma
Acinar cancer
Tubular scirrhous cancer
Paget's disease
Tubular carcinoma
Alveolar carcinoma
Cystic fibroma
Reclus' disease
Abnormal involution
Senile parenchymatous hypertrophy
Solid fibroma
Schimmelbush's disease
Cystic sarcoma
Solid sarcoma

In regard to our knowledge of carcinoma one finds two main groups of writers expressing themselves on its etiology, which is often confused with histogenesis. One group attributes the epithelial perversion to changes which take place primarily in the connective tissue stroma of the breast. This primary change allows overgrowth and invasion by the epithelium. This theory has or has had many advocates, i. e., Mintz (12), Ribbert (13), Lichtenhahn (14), Koenig (1) and others. The last named investigator and perhaps others have departed from this belief.

Tietze (15), Saar (16), Schimmelbusch (17), Brissaud (18), Sicre (19), and Reclus (20), have strongly advocated the primary change to be in the epithelium itself and not in the connective tissue.

There are still authorities who believe that the condition of carcinoma is the result of proliferation of both stroma and epithelium—Lichtenhahn (14), Koenig (1), Maly (21), Roloff (22), Stiles (23), and others.

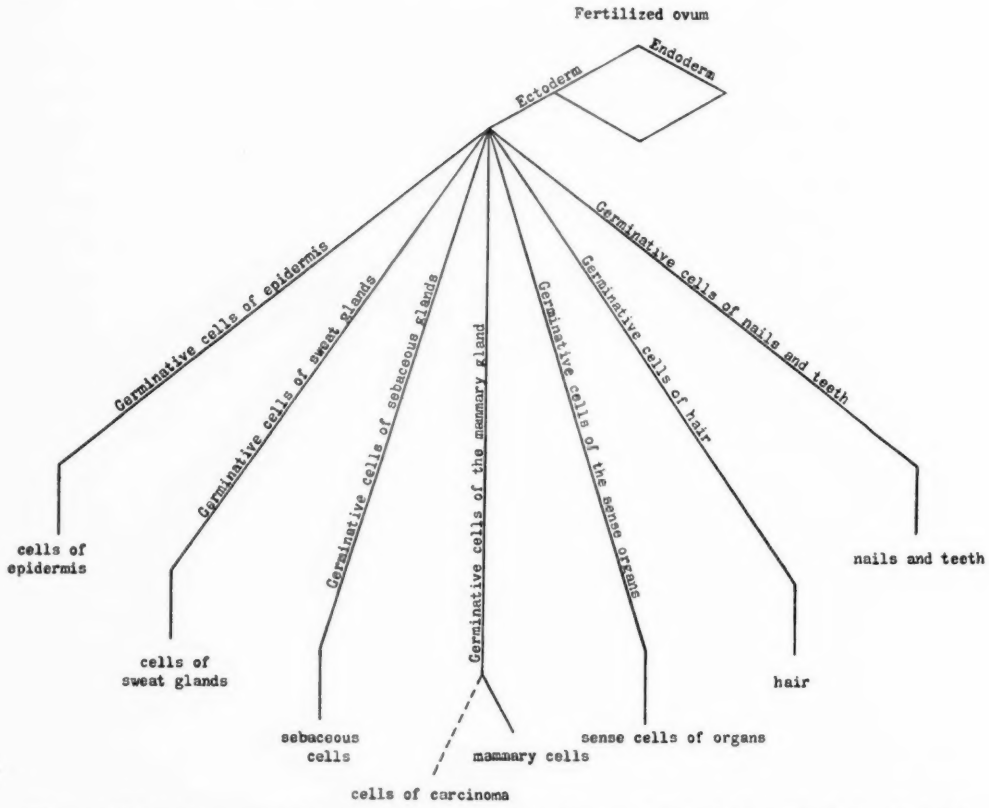
The theory of "cut off epithelium" or "epithelial embryonic rests" is very strongly fixed in the minds of the practitioner and older teachers as the origin of neoplasmata.

Many authorities believe that carcinoma may develop on a "cystadenoma" or a "chronic mastitis." This belief has been expressed by Keibel (24), Tietze (15), Kuru (25), Bobbio (26), Fremicourt (27), Brehm (28), de Quervain (29), Verga (30), Sicre (19), Schimmelbusch (17), Reclus (20), Roloff (22), Franko (31), Sasse (32), and Koenig (1).

This suspicion remains, however, to be substantiated by fact. Nobody has ever seen such actual development take place. One may, however, scientifically state that carcinoma is seen in association with these conditions.

The "tumor process" or "neoplastic process" in the breast has been insinuated by Schimmelbusch (17), Saar (16), Franko (31), Maly (21), and others.

The interpolation of authors' names in this brief discussion of literature is made with the keen understanding that writers often change their opinions soon after the publication of their articles. They are mentioned here simply to emphasize the fact that the exact nature of the process is unknown. Writers, however, have continued to express themselves sufficiently emphatically to students to have staunch followers. Most of the views



Diagram—Schematic representation of the relation of the ectoderm to the germinative cells of the skin and its appendages and the apparent biologic position of the cells which comprise carcinoma of the breast.

of the subject have been derived from a study of a limited material with limited impressions or perhaps to present a working hypothesis.

The limitation of the observed, described, published and accepted material may be seen from the fact that each of eight out of eleven authorities who are frequently quoted drew and published conclusions from less than five cases.

Two authorities, whose names are attached to indefinite pathologic conditions [1] in the breast, arrived at such eminence by describing a very small series of cases. Both articles were written, however, earlier than 1890 — a fact which should make critics and future writers extremely charitable. Material was not so abundant and microscopic pathology was not so well developed at that period.

A large number (758) of cases was published by a surgeon (Warren, 2). Pathologic reports were utilized instead of actual research on each specimen. Definitely diagnosticated groups of apparent entities were utilized from which to analize clinical and prognostic data.

With this brief critical summary of methods of investigation, record and teaching of the pathologic conditions in the breast, one may proceed to a consideration of facts relative to the material at hand.

The material under consideration during this study in the Mayo Clinic consisted of

[1] "Schimmelbusch's Disease" and "Reclus' Disease."

1000 amputated or excised human breasts for various pathologic conditions.

In this organ the so-called benign and malignant neoplasms and inflammatory conditions require removal and therefore supply a wide field for the investigation of epithelial cells under many conditions. It supplies solid and cystic benign and malignant conditions. The epithelium is especially simple, beautiful and definitely arranged under normal conditions.

The methods of investigation consisted of:

1. Studying the perfectly fresh specimens [1] both macroscopically and microscopically.

2. Repeated microscopic examinations of all portions of the organ by means of fixed sections stained with hæmotoxylin and eosin.

3. Large numbers of gross and microphotographs which, when studied collectively, give a comparatively complete picture of the neoplastic pathology of the organ. These pictures arranged themselves naturally according to similarity or identity and formed a series which begins with the simplest conditions of the units [2] (acini) of the organ and ends with the most extensive destruction of normal structure.

4. Repeated examinations of the material in order to confirm the natural photographic arrangement of the pathologic processes existing in and about the epithelium.

The embryological or developmental fatcs, which are correlated with the writer's findings in this paper, are well known to embryologists and may be investigated by studying the works which are appended to this paper. If one traces the life-history of the mammary epithelium one begins with the ectoderm of the three layer stage of embryologic development. This layer of partially differentiated epithelium becomes more highly developed to form the so-called skin of the embryo. At first it is composed of one layer of low cuboidal cells, which, with further development of the embryo becomes differentiated to form two or more layers of cells (Figs. 1 and 2) — the outer layers differing from the first layer in being flatter or less cuboidal with their long axes parallel with the surface of the body (Fig. 1).

With both antenatal and postnatal development the secondary layers become more differentiated and less like their immediate predecessors which, in this stage, the embryologists have termed the "stratum germinativum" [3] (Figs. 1 and 2)[4] of the skin or the germinating layer of the epidermis.

The history of the cells of this layer proves that they retain the power of divergence into several structural and functional derivatives. It may be spoken of as a plastic layer at least in the embryo. This expression of its broad functional capacity is based on its behavior in the development of the appendages of the skin; namely, hair, nails, sweat glands, sebaceous glands and the mammary gland.

Histologic specimens which have been taken through the embryonic skin and subcutaneous tissue in various portions show developmental activities of the stratum germinativum other than the production of epidermal cells.

Figs. 4 to 15 inclusive show evidence of activity directed toward the development of hair follicles. The downward growth of the cells of the "stratum germinativum" into the subcutaneous tissue is accompanied by their differentiation into hair instead of epidermis. The examination of a fully developed hair follicle shows that there still remains a row of cells (Figs. 8 to 10 inclusive) which correspond to the "stratum germinativum" of the epidermis and may be considered the stratum germinativum of the hair. The epithelium of nails, sebaceous glands (Fig. 16), sweat glands (Figs. 11 to 15 inclusive), and mammary glands (Figs. 17, 18, 19) possesses its stratum germinativum from which its differentiated cells are reformed.

From this brief statement of embryologic facts and the photographic facts which are presented here one may crudely construct a working diagram of the probable histo-

[1] All specimens were received into the surgical laboratory immediately upon removal. Frozen sections were made from many areas and stained with Unna's polychrome methylene blue.

[2] The breast is composed of adipose tissue, connective tissue, and epithelial tissue. The last of these, or secreting portion of the breast, is arranged anatomically into gland groups which are composed of from 6 to 15 gland units. The gland unit or secreting unit represents the histological unit of the organ and consists of several cells sharply demarcated from the stroma and arranged around a space or lumen which may be potential or patent.

[3] Synonyms: stratum germinativum, stratum cylindricum, basal epithelial layer.

[4] Illustrations and legends Figs. 1 to 10 are taken from Kiebel & Mall's Embryology.

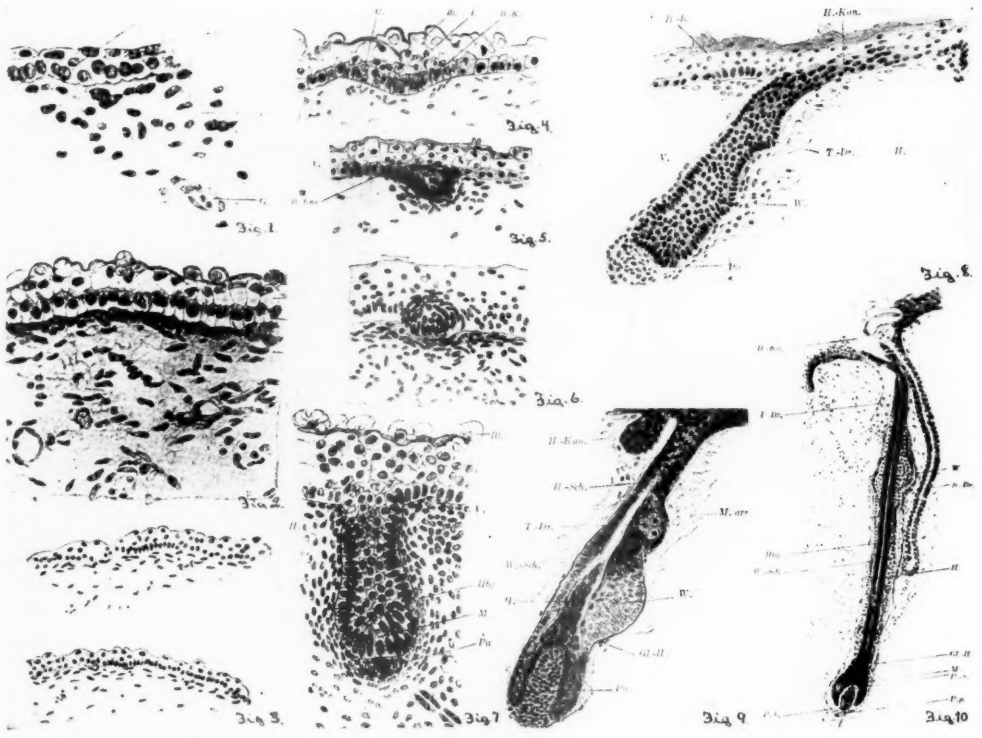


Fig. 1. "Human fœtus 32 mm. in greatest length (collection of Prof. Robert Meyer, No. 307). Integument from the right side of the body; the epidermis is beginning to become three-layered. P, periderm; L, stratum intermedium; B, stratum germinativum; C, corium, rich in cells; G, vessel. x400."

Fig. 2. "Human fœtus, 85 mm. vertex-breech length, male. Epidermis distinctly three-layered. P, periderm with partly separated cells; I, stratum intermedium; B, basal layer of cells with mitosis; C, corium, rich in cells (mostly spindle-shaped). Mammary region. x430."

Fig. 3. "Primary hair germ of human fœtus, 8.5 cm. in length, male, from the mammary region. Two successive sections in which the circumscribed approximation of the nuclei is visible. The cells of the stratum intermedium have not yet increased in number and there is no increase of the connective-tissue cells of the corium. x41."

Fig. 4. "Hair germ from the same region as Fig. 3. H.-K., hair germ; I, increase of the cells of the stratum intermedium, and C., of the connective-tissue cells of the corium; Bl, periderm cell. x430."

Fig. 5. "Longitudinal section of a hair germ of another 8.5 cm. human fœtus, more advanced stage. H.-Kan., hair canal cells (Stöhr). Spaces exist among cells of germ. V, anterior surface; H, posterior surface hair germ. x430."

Fig. 6. "Transverse section of a hair germ in the same stage as Fig. 5, from pectoral region of the fœtus of Fig. 3 and 4. Shows the distinctly symmetrical structure and the kiln-like arrangement of the cells. x430."

Fig. 7. "Hair papilla from the upper lip of the fœtus of Figs. 3, 4 and 6. M., hair matrix with spaces at the converging point of the matrix cells; Pa., anlage of papilla; Hbg., connective-tissue hair sheath; Bl., vesicular cells of the periderm; V, anterior surface of the follicle; H, its posterior surface with the (scarcely recognizable) differentiated arrangement of the nuclei (sebaceous gland and region of the hair swelling). x430."

Fig. 8. "Hair papilla in a more advanced stage, transition into the bulb papilla. Pa., anlage of papilla; W, swelling; T.-Dr., anlage of sebaceous gland; H.-Kan., hair canal cells; H.-K., tangential section of a neighboring hair germ. x230." (After Stöhr: Lehrbuch der Histologie, Fig. 300; Entwicklung des menschlichen Wollhaares, Fig. 9).

Fig. 9. "Sheath hair still near the bulb-papilla stage. Pa., papilla; Gl.-H, vitreous layer; W, swelling; M. arr., musculus arrector pili; H.-Sch., inner root sheath; T.-Dr. sebaceous gland; W.-Sch., outer root sheath; H., cells of the hair cone, not yet differentiated. x230." (After Stöhr: Lehrbuch der Histologie, Fig. 302; Entwicklung des menschlichen Wollhaares, Fig. 14).

Fig. 10. "A completely formed lanugo hair, from the mammary region of an eight-months' male fœtus. Partly schematic, reconstructed with the camera lucida from an oblique series of sections. P.p., cushion of connective-tissue papilla; P.h., neck of papilla; P.-S., tip of papilla; M., hair matrix; Gl.-H., vitreous layer, for the most part separated from the outer root sheath; H., shaft of hair; W.-Sch., outer root sheath; Hbg., connective-tissue portion of follicle; S.D., sudoriparous gland; W., swelling; T.-Dr., sebaceous gland; H.-Ka., hair canal. x120."

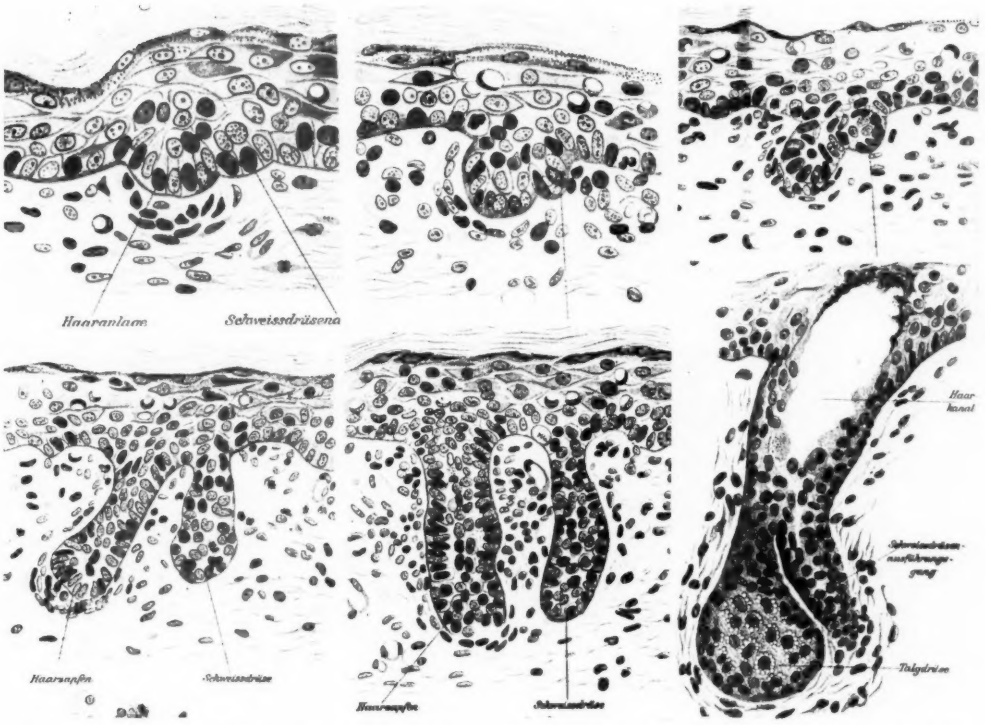


Fig. 11. The formation of the "anlagen" of a hair follicle and a sweat gland from the stratum germinativum. (Taken from Fig. 38, C. Wimpfheimer, Anat. Hefte I Abteilung, 104.)

Figs. 12, 13, 14 and 15 show the further development of the "anlagen" of the hair follicle and a sweat gland (Fig. 11). (C. Wimpfheimer.)

Fig. 16 shows the relation of the "anlage" of sebaceous glands to the epithelium of the skin. (C. Wimpfheimer).

genetic position of the cells which form carcinoma.

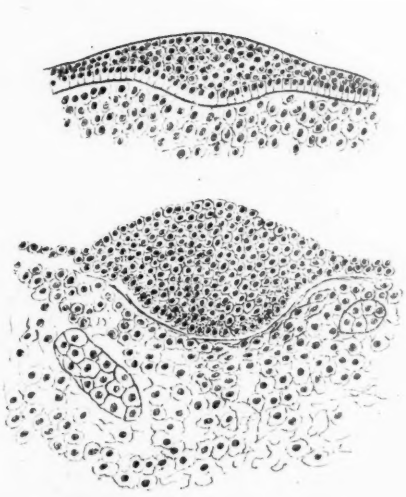
Figs.[1] 20 (A27943),[2] 21 (A8743) and 22 (A27943) were taken from sections of adenofibromata and non-carcinomatous portions of carcinomatous breasts. The acini vary greatly in size, some are atrophic, some cystic and some hypertrophic. They are separated by a fairly dense irregular interacinar stroma. A few lymphocytes are seen in the field. These pictures correspond to many of the pictures which are published in the literature as "abnormal involution," "senile parenchymatous hypertrophy," "Schimmelbusch's disease," "Maladie de Reclus" and "mastitis chronica cystica." Such pictures are almost always present in chronic cystic mastitis.

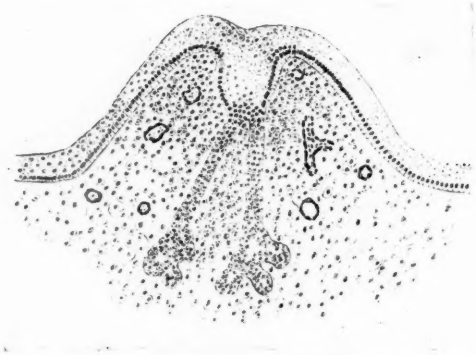
In Figs. 23 (38463), 24 (A27943), 25 (26079), and 26 (26079) one sees higher magnifications of acini similar to those which are seen in Figs. 20, 21 and 22. In all, each acinus is composed of two rows of epithelial cells, the two rows being more prominent in Figs. 26 and 27. The cells are evidently hypertrophic and hyperplastic. Those lying

[1] Pathological diagnoses of cases utilized to illustrate this paper.

A27943	Fibro-adenoma	31329	Carcinoma
A8743	Fibro-adenoma	30694	Carcinoma
26079	Extensive carcinoma	A41253	Carcinoma
		26166	Carcinoma
36138	Carcinoma	50132	Carcinoma
28376	Carcinoma	38463	Carcinoma
36116	Carcinoma	20797	Fibro-adenoma
A12998	Chronic mastitis	A74345	Carcinoma
35256	Carcinoma	29785	Carcinoma
22595	Carcinoma		

[2] Numbers in parentheses preceded by A are Office numbers. Those without the A are Hospital numbers.





Figs. 17, 18, 19 present the development of the mammary gland from the stratum germinativum of the skin. (Copied from illustrations in the "Lehrbuch der Entwicklungsgeschichten von Dr. Robert Bonnet.)

next to the lumen are more deeply stained, have dense protoplasm and oval nuclei. They are apparently more highly differentiated than those of the outer row and are the secreting cells of the acinus. The cells of the outer row have clear, rare protoplasm; the nuclei are round; the cells are less differentiated; they are more embryonic. They form the "stratum germinativum" for the secreting cells of the acini.

In Fig. 28 (36138) the inner row of differentiated cells has completely disappeared, leaving only the less differentiated cells of the outer row. The acini are surrounded by active, proliferating connective tissue cells.

In Fig. 29 (28376) the cells of the outer row or the undifferentiated cells are hyperplastic and have filled the entire lumen. None of the cells of the inner row are to be seen.

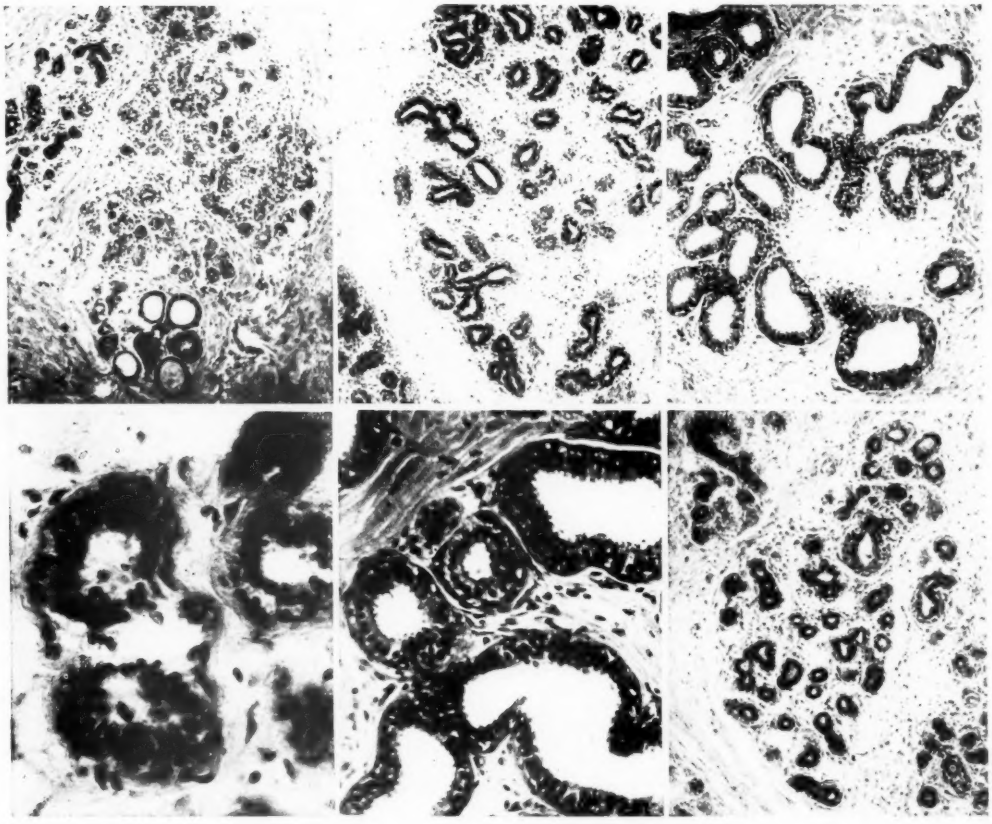
In Fig. 30 (36116) the acinus is formed by hyperplastic cells of the outer row. They are large and irregular in shape and size. In the center of the acinus the detritus of disintegrated cells is seen. The periacinar connective tissue is composed of active fibroblasts.

The acini in Fig. 31 (12998) are filled with disintegrating hyperplastic cells of the outer row. This picture corresponds to some of those published in the literature as "Schimmelbusch's disease," "senile parenchymatous hypertrophy," "Maladie de Reclus," and "mastitis chronica cystica." It will be noted that the periacinar connective tissue is dense. It is not filled with lymphocytes or proliferating fibroblasts. This association of dense connective tissue with complete necrosis of the cells of the acinus may be seen also more distinctly in Figs. 64 to 67 in which there is formed a definite barrier of connective tissue surrounding the acinus.

Figs. 32 (36116) and 33 (36116) present acini which are composed of only one row of cells which correspond to the cells of the outer row of undifferentiated cells. They are hypertrophic, hyperplastic, and exceed the normal limits of size and shape of the cells of the outer row. In the center of the acinus one finds disintegrated material, which, when studied in relation to the foregoing pictures, is probably disintegrated epithelium.

Fig. 34 (36116) shows a similar acinus with a break in the acinar wall.

Fig. 35 (35256), which is a lower power photograph, shows a number of acini which answer the descriptions given for Figs. 26, 27, 28, 29 and 30, and show clearly the great distortion of the acinar outlines with confusion of cells between the stroma and the epithelium.



Figs. 20 (A27943), 21 (8743), 22 (A27943), 23 (28463), 24 (A27943), 25 (26079). Sections taken from adeno-fibromata and non-carcinomatous portions of the mammary gland. The pictures are similar to some of those which have been published under the heading of "abnormal involution" (Warren), and described by Koenig as "mastitis chronica cystica." Epithelium in the stage of *primary hyperplasia*.

Fig. 36 (36116) presents a picture of hyperplastic epithelium frequently seen in "chronic cystic mastitis" and practically always in association with definite invasion of tissue.

Figs. 37 (31329), 38 (31329), 39 (31329), 40 (30694) and 41 (22595) show the morphologic similarity of the extra-acinar cells (carcinoma cells) to the intra-acinar cells which have been seen in the acini in the preceding pictures. They are definitely carcinoma and present the cellular characteristics which are present in cells which have not "broken through" the so-called "basement membrane," — the accepted morphologic criterion of malignancy.

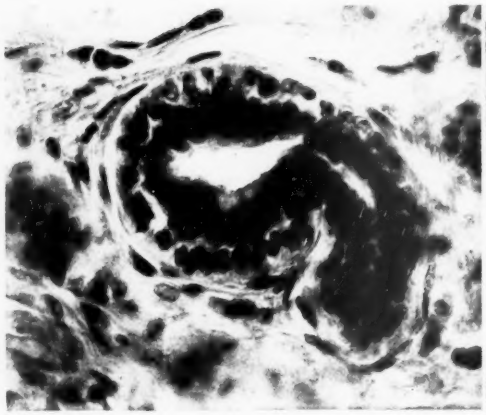
Figs. 42 (A41253), 43 (A41253), 44 (26166), and 45 (26166) show extensive invasion of tissue by perverted epithelium.

The morphologic characteristics of the epithelium which lines mammary cysts is similar to that which has just been described.

Fig. 46 (A27943) is a section through a specimen of chronic cystic mastitis showing small cystic acini.

Fig. 47 (50132) is a photograph of fresh tissue showing interacinar bridges in one stage of the process of destruction.

In Fig. 48 (35256) the acini of a gland group may be seen in the process of fusion to form cystic areas.



Figs. 26 (26079), 27 (36138). Acini from a non-carcinomatous portion of a carcinomatous breast. The cells of the acini form two distinct rows (inner and outer rows). This condition is common in chronic cystic mastitis, "abnormal involution," "senile parenchymatous hypertrophy," "Schimmelbusch's disease," "Maladie de Reclus," cystadenoma and so-called benign fibro-epithelial neoplasms. Under the heading of hyperplasia it is considered *primary hyperplasia.*

Figs. 49 (12998) and 50 (29785) show acini which have become united and form pseudopapillæ which have been covered over with epithelium, which is arranged in two definite layers comparable to the arrangement which is seen in the acini in Figs. 26 and 27.

In Figs. 51 (38463) and 52 (38463) a distended cyst is seen. It is lined by the same two layers of epithelium which are found in Fig. 26.

In Fig. 53 (12998) and 54 (12998) one finds distended acini which are a part of a gland group composed of 7 acini. Unlike the cyst which has been seen in Fig. 51, there is only one layer of epithelium lining most of the wall. There is, however, an extensive hyperplasia of the cells similar to that seen in Figs. 28 to 36 inclusive.

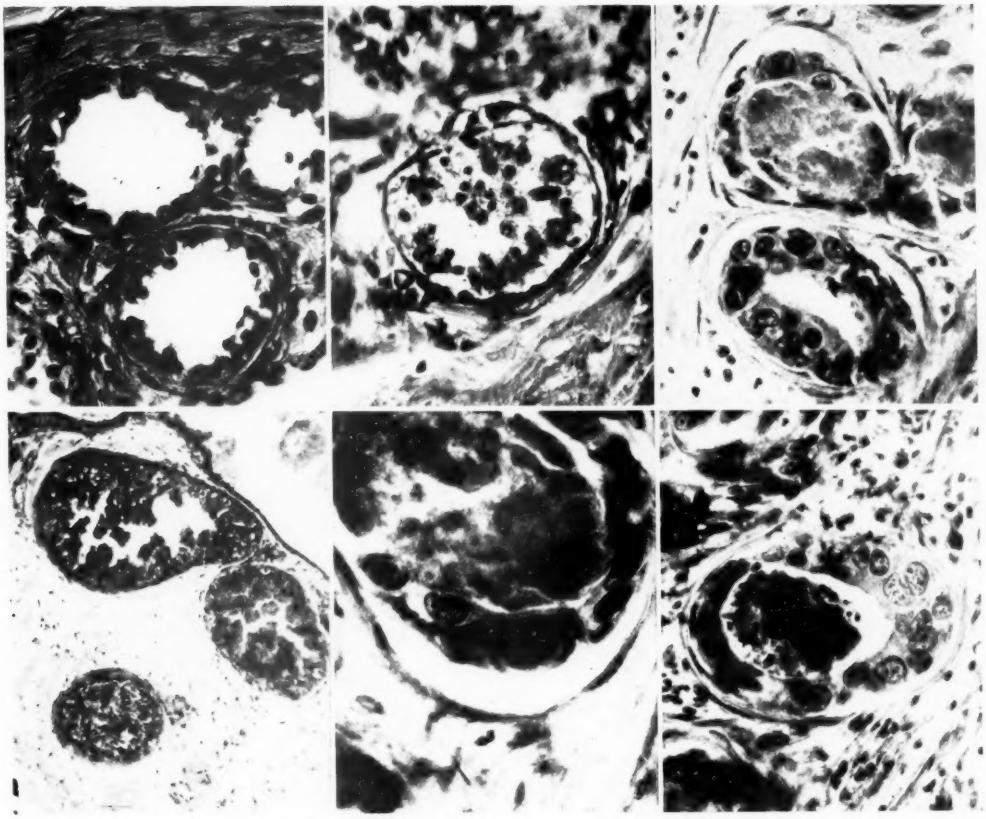
In Fig. 52 (38463) may be seen the character of the cells which arrange themselves in two rows to line simple cysts. This picture may be compared to Figs. 55 (36116), 56 (20797), and 57 (36116) in which the inner row of cells has completely disappeared and is replaced by hyperplastic cells of the outer row. The nuclei of the cells are irregular in size and shape beyond the normal limits of size and shape. Many of the cells are apparently necrotic. The line of demarcation between the epithelium lining the cyst and the periacinar stroma is sharp. A few lymphocytes are seen in the stroma.

In Figs. 56 (20797) and 54 (12998) one finds hyperplastic cells lining the cyst. The nuclei are irregular in shape and size. The line of demarcation is less sharply defined. In Fig. 57 (36116) the line of demarcation is still less defined, the epithelial cells are hyperplastic and very irregular in shape and size.

Fig. 58 (A74345) with its accompanying small pictures (59, 60, 61, 62 and 63) is important in showing the variety of epithelial cells which may be present in the same field of the microscope. Two cysts are present; one is lined by several layers of small oval and round epithelial cells. The second cyst is lined by low cuboidal cells and columnar cells. Near the cysts we see acini filled with large cells with small, round nuclei. These cells are indistinguishable from the cells which one finds characteristic of sebaceous glands.

The last picture is frequently found in the breast and doubtless has been responsible for the speculation of a few observers who have written about the possibility of carcinoma of the breast arising as a result of misplaced sebaceous glandular elements.

The photographs herewith presented represent a composite picture of pathologic



Figs. 28 (36138), 29 (28376), 30 (36116), 31 (12098), 32 (36116), 33 (36116). *Secondary hyperplasia* in sections from chronic mastitis and carcinomatous breasts. The inner row of cells has disappeared and the outer row has become hyperplastic. The cells are indistinguishable from the carcinomatous cells in Figs. 37, 38, 39, 40 and 41.

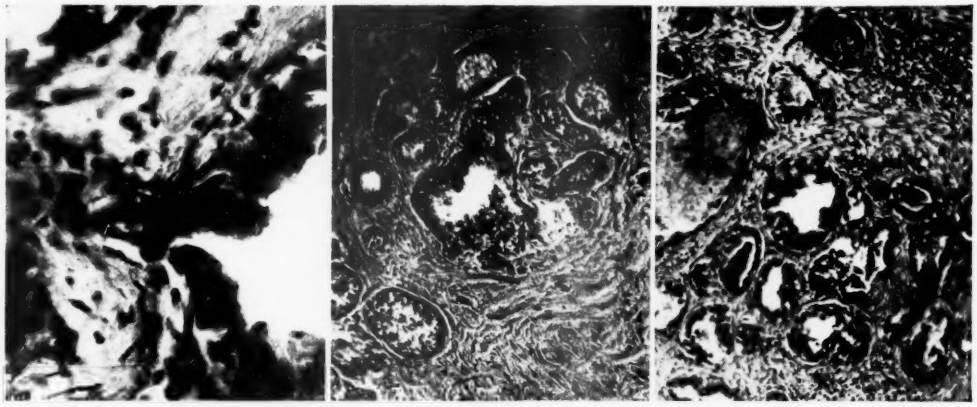
conditions in the breast other than sarcoma, chondroma, tuberculosis, myxomata and such rarer conditions as actinomycosis, and abscesses, and are clearly those found in chronic cystic mastitis and carcinoma. Similar epithelial hyperplastic conditions are also seen in encapsulated tumors such as adeno-fibromata, fibro-adenomata, and intracanalicular fibromata.

The usual behavior of the epithelium of the acini under conditions of chronic inflammation — without any reference whatsoever to the cause of the condition — is herewith seen photographically. The behavior of the epithelium of the tubuli will be reserved for a future paper.

The breast, as seen in operative material, frequently shows evidence of chronic inflammation which is indicated by varied amounts of lymphocytic infiltration, connective tissue and epithelial proliferation, acinar distortion, epithelial necrosis, exfoliation and hyaline degeneration.

The pictures which have been described and published in the literature under the titles of "chronic cystic mastitis," "abnormal involution," senile parenchymatous hypertrophy" and "Schimmelbusch's disease" are similar to the pictures which are presented in Figs. 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 31, 47, 48, 49, 51, 52, 53, 54, 55, and 56.

Where to draw the line between the hyper-



Figs. 34 (36116), 35 (35256), 36 (36116). *Tertiary hyperplasia.* The cells are apparently similar to those in "secondary hyperplasia." The line of demarcation between the parenchyma and the stroma is indefinite or partially destroyed.

plastic changes of chronic cystic mastitis and the pictures which are definitely carcinoma is beyond the writer's present power. A careful study of the illustrations shows that there is no apparent sharp line of demarcation. One condition merges into the other.

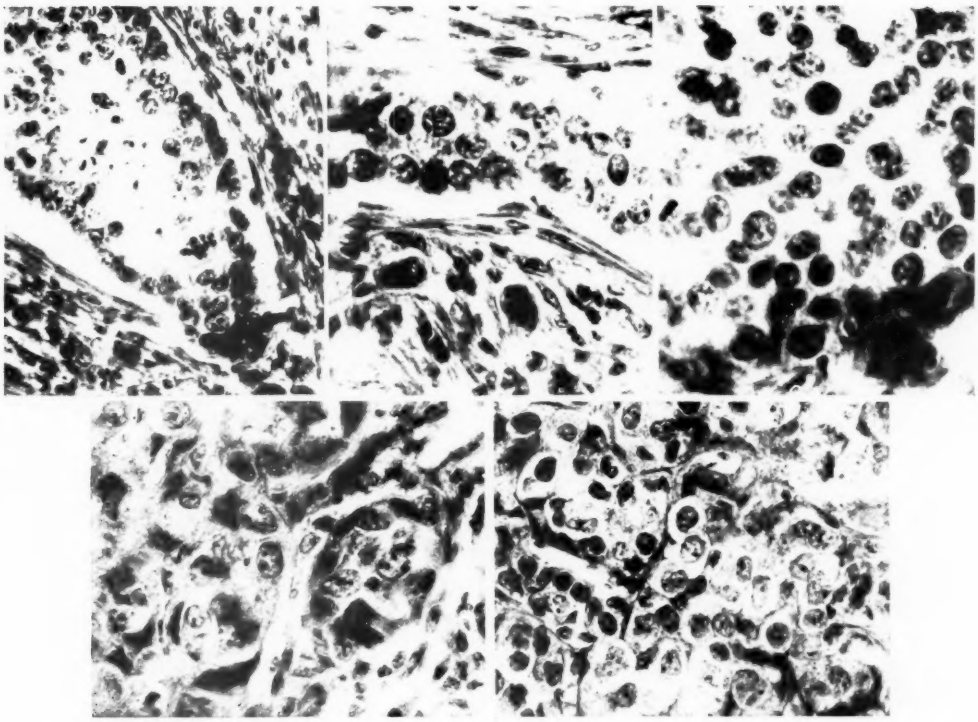
Why all cases of chronic cystic mastitis are not associated with carcinoma is no more answerable than why the acute bacterial diseases do not terminate fatally in all cases and why all cases do not carry out their course in exactly the same manner and with the same amount of destruction. Bodily resistance, whatever that means, doubtless plays a rôle in the progress of epithelial neoplasia just as in the case of any other pathologic conditions. The part played by the stroma in epithelial hyperplasia and neoplasms varies considerably as is seen in Figs. 64 (36116), 65 (29785), 66 (36116), and 67 (36138).

The question of why and how this defensive warfare is carried on in the body is one of the fields of pathology and physiology which have been barely touched by the scientific world.

So far as the histogenesis of carcinoma is concerned in the breast one may say that not infrequently, usually between lactations or shortly after the menopause, certain changes of a chronic inflammatory nature occur and that associated with these changes one finds an hyperplasia of the epithelium of the acini, characterized by prominence of the outer row of cells (stratum germinativum) and that frequently in one or more portions of the breast the inner row or secreting cells are exfoliated and not renewed by the outer row. Instead of renewal of functional or differentiated cells, the stratum germinativum continues to proliferate into the acinar lumen in an undifferentiated condition.

In this condition of the acini the interacinar septa often become thinner and disappear, thereby producing a cyst by coalescence. This coalescence occurs with and without the presence of the two layers of epithelium. In the presence of the hyperplasia of the outer row the line of demarcation between parenchyma and stroma frequently is confused and is often absent with invasion of the periacinar stroma by the same cells, or at least cells indistinguishable from the cells of the outer row. This last condition produces the histologic picture which is recognized carcinoma, which clinically is almost always if not always malignant.

An analysis of epithelial hyperplasia and neoplasia in the light of the development of epithelium of the breast may lead some investigator to determine more accurately the exact biologic position of the so-called carcinomatous cells which we know are contrary



Figs. 37 (31329), 38 (31329), 39 (31329), 40 (30694), 41 (22595). *Tertiary hyperplasia* (migratory hyperplasia) showing morphologic characteristics of the cells of carcinoma in the stroma of the breast.

in action to the activities of normal cells under a communistic life.

In the terms of hyperplasia three grades may be recognized in the breast. These are the morphologic extremes of an apparent process of transition, the transitional stages of which supply no distinctive terminology.

Under the head of *primary hyperplasia* one may group all of the hyperplastic epithelial morphologic evidences of activity which pertain to the combined activities of both the differentiated cells and the undifferentiated or germinal cells. All conditions in which both rows of cells exist and partake in the reactions may be spoken of as primary hyperplasia.

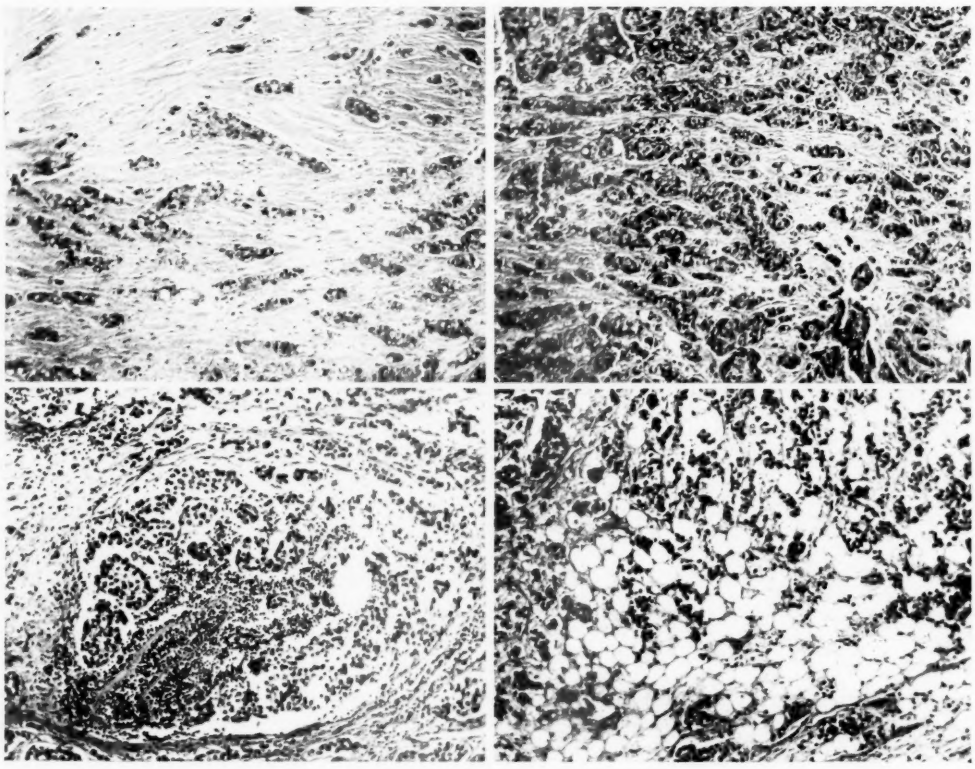
Under the head of *secondary hyperplasia* one sees only the activity ot the germinative or undifferentiated cells within the confines of the acinar limits. They have failed to produce the differentiated cell.

A third grade — *tertiary hyperplasia* — is recognizable in the extra-acinar or migratory activities of the cells which apparently originated in the germinative cells (cells of the outer row [1]).

The question which immediately rises on the presentation of such a simple conception of hyperplasia — of which carcinoma is here considered a part — is, What clinical and prognostic deductions may be drawn?

This question with our present knowledge cannot be momentarily answered any more than the clinician can accurately prognosticate according to the terminology of the so-called "types" of carcinoma which he utilizes at present. The favorite question of the clinician is, What kind of cell constitutes

[1] Stratum germinativum of the mammary epithelium.



Figs. 42 (A41253), 43 (A41253), 44 (26166), 45 (26166). Tertiary hyperplasia (migratory hyperplasia) showing low power morphology of the stroma which is invaded by carcinomatous cells.

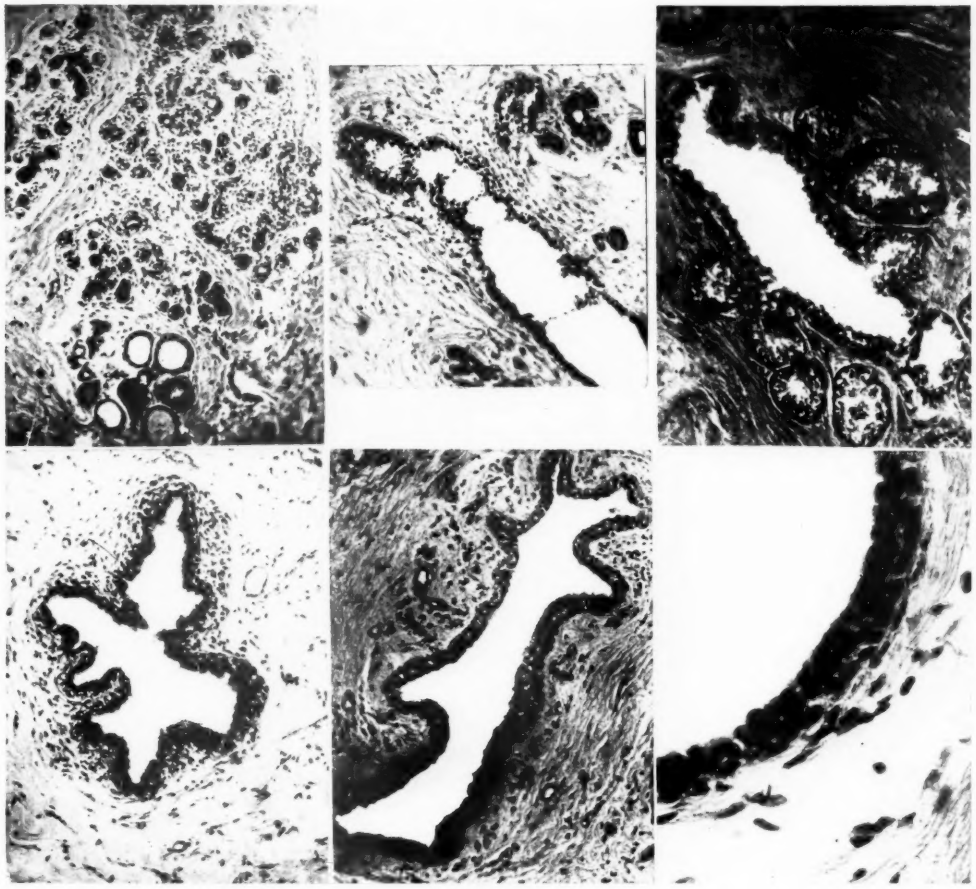
the neoplasm? In the writer's experience only two apparent extremes even suggest prognostic difference, namely, the so-called "medullary or cellular carcinoma" and the "scirrhus carcinoma." Intermediate gradations between these so-called entities exist and often both "types" exist in the same tumor—indeed, in the same field of the microscope.

Again, in the summation of our real knowledge it seems that we are forced to admit, when not deceived by the suggestion of empiricism, that accurate knowledge of the activities, the duration of activities of epithelium and the bodily defensive forces against malignant invasion, is still in the realm of research.

Such a statement of facts may seem pessimistic. On the contrary, however, they represent merely the "taking stock" of things as they are and not as we are prone to think they are in our partially empirical efforts to accomplish things.

So far as the epithelial activities in the breast are concerned with the therapeutic efforts of the clinician, the present conception offers him a greater chance of doing more good when his efforts are controlled by the activities of those specially trained in the recognition of the stages herewith presented.

It immediately gives him a logical grouping for his material, a grouping which is based on definite morphologic and easily recognizable conditions. It presents three distinct conditions which can be studied in conjunction with good pre-operative and post-operative



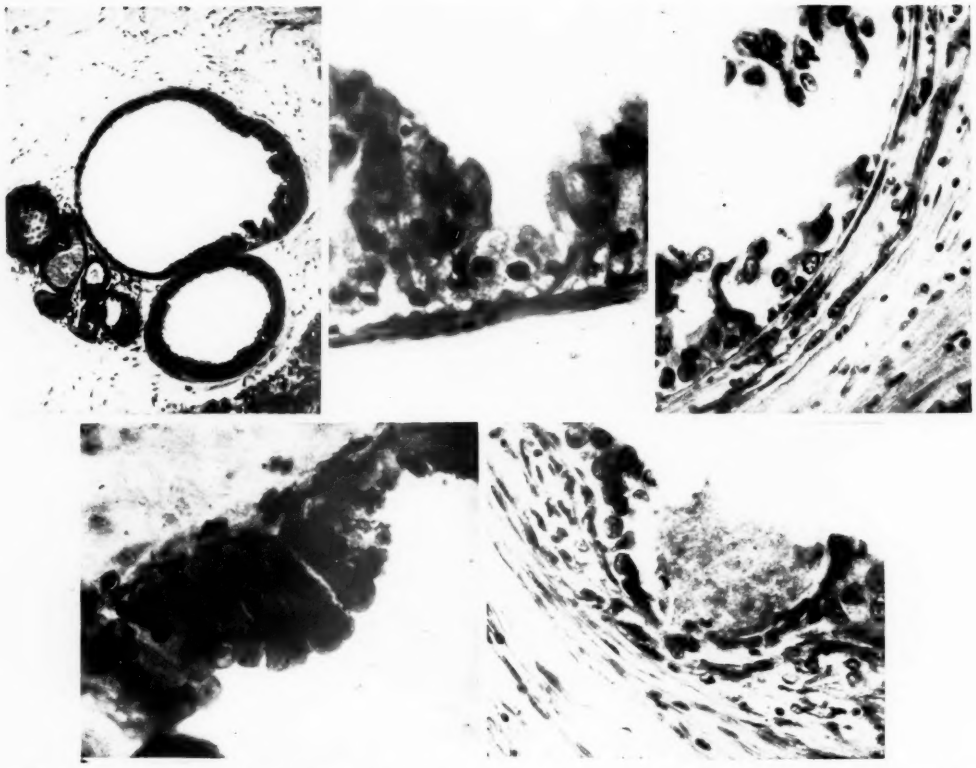
Figs. 46 (A27943), 47 (50123), 48 (35256), 49 (12908), 50 (29785), 51 (38463), 52 (38463) show the evolution of at least some of the cysts which occur in the mammary gland. The stages are apparently dilation of acini, fusion of acini, formation of pseudo-papillæ, further distention with disappearance of the pseudo-papillæ. The epithelium is in the two-layered stage of hyperplasia (primary hyperplasia).

histories. The morphologic entities are not based on such an indefinite histologic structure as the "membrana propria" which is only occasionally demonstrable even under normal conditions.

On a careful study of the pictures presented here, which represent the findings in a large series of cases, the following deductions may — it seems to the writer — be made:

It may readily be seen that there is a definite pathologic foundation for the surgical experience that the conditions described

Fig. 52.



Figs. 53 (12998), 54 (12998), 55 (36116), 56 (20797), 57 (36116) show secondary hyperplasia of the epithelium of cysts in carcinomatous breasts. The inner row of epithelial cells has disappeared, the outer row is hyperplastic and the cells themselves are indistinguishable from those which are seen in Figs. 28 to 41.

as "senile parenchymatous hypertrophy," "abnormal involution," "cystadenoma," and "chronic cystic mastitis" are often, if not always, associated with carcinoma.

In a single specimen may be seen the stages through which this association probably takes place.

One also finds histologic pictures in specimens of carcinoma which correspond to those described by many writers as "cystadenoma," "papillary cystadenoma," "intra-cystic epithelioma," "Schimmelbusch's disease," "chronic interstitial mastitis," "chronic cirrhosing mastitis," "chronic cystic mastitis," and "polycystic epithelial mastitis."

Carcinoma is apparently an outgrowth of the outer row of cells of the acini and not of the secreting cells, and also is not the product of "cut off" epithelial cells by scar tissue, but is derived from the stratum germinativum.

The cells of the outer row of the acini form the growth into the lumen described by Schimmelbusch as cystadenoma. The proliferating cells of the acini — whether these acini are cystic or not after the disappearance of the inner row of cells — present the same irregularities of the nuclei which are seen in the cells which have invaded the stroma, lymph spaces, and lymph nodes. They are irregular in size and shape beyond the normal limits of size and shape of the normal cell. This irregularity is present in the acini, the so-called "basement membrane" of which is still intact.

The practical question, therefore, arises:

Fig. 58.

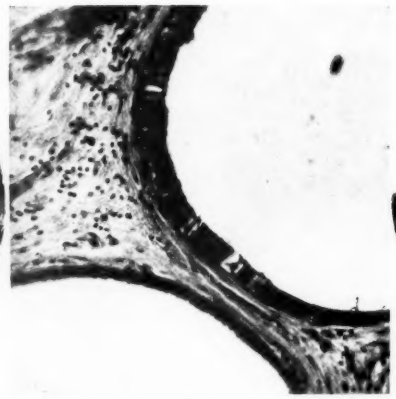


Fig. 59.

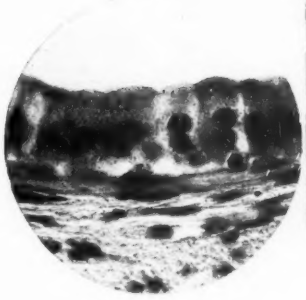
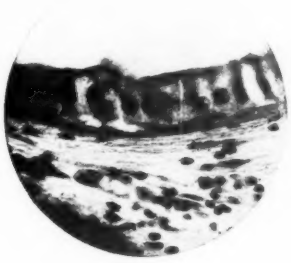
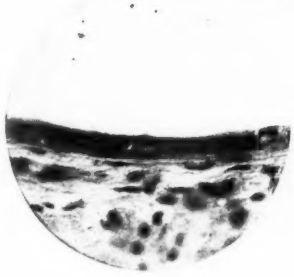


Fig. 60.





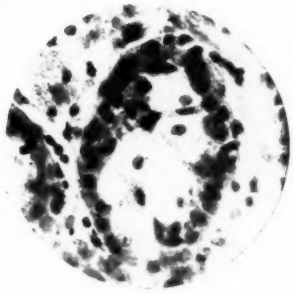


Fig. 61. Fig. 62. Fig. 63.

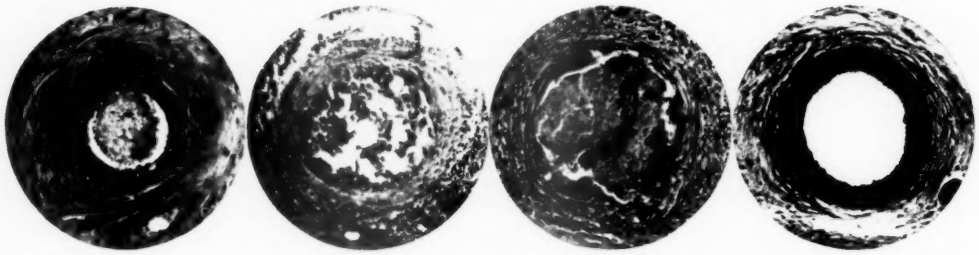
Fig. 58 (74345) shows a single microscopic field through a non-carcinomatous portion of a carcinomatous breast. One sees that four distinct "types" of cells are present. Small round or flattened cells arranged in two rows, cuboidal cells, columnar cells and cells which are morphologically indistinguishable from the cells of a sebaceous gland.

Figs. 59, 60, 61, 62, 63. Higher magnifications of the different types of epithelium in Fig. 58 (74345).

Is it necessary to wait for the penetration of the "basement membrane" before making a diagnosis of carcinoma? From the standpoint of the general pathologist this may be necessary, but if the surgeon waits for such a change he will be basing his procedure upon a definition of carcinoma which does not consider the same characteristic in the cells before and after the penetration of the "basement membrane" distinctive of carcinoma. If the pathologist considers the penetration of the "basement membrane" the essential characteristic of carcinoma, then it must be admitted that the cells in chronic mastitis and cystadenoma are often just as irregular as in carcinoma and are indistinguishable from those of carcinoma.

A more careful and detailed study of carcinoma cells, "cystadenoma" cells, the hyperplastic epithelial cells of fibro-adenoma and of chronic cystic mastitis has demonstrated that the resemblance between these cells is so great that if the desired therapeutic effect is to be accomplished, more "benign" tumors (fibro-adenoma, adeno-fibroma, and cystadenoma) must be completely enucleated and more breasts diagnosticated as "chronic cystic mastitis," "abnormal involution," or "senile parenchymatous hypertrophy" must be completely removed.

On account of the close morphologic relation between these conditions the diagnosis must be made and the therapeutic measures carried out early in order to save the patient



Figs. 64 (36116), 65 (29785), 66 (36116) and 67 (36138) show the periacinar reaction which often occurs in association with carcinoma.

from the ravages of the extensive hyperplastic migratory epithelial conditions which we call carcinoma.

Gross diagnosis, even when made by an expert, can be made in only a certain percentage of cases. The early change is microscopic, especially in fibro-adenomata and chronic cystic mastitis.

Early diagnosis must be carried out regardless of the age of the patient, because carcinoma occurs in a wide range of ages. The youngest of this series was aged 23 years, the eldest 72 years and the average age was 47 years. The youngest "benign" fibro-epithelial tumor occurred at 19 years, the eldest at 65, and the average 33 years.

The surgeon as well as the pathologist should look upon the histogenesis of carcinoma as a process which apparently bears a most striking relationship to circumscribed and diffuse cystic or non-cystic fibro-epithelial hypertrophies and hyperplasias of the breast.

With our present knowledge of conditions of the mammary epithelium it behooves the pathologist and the clinician to accept a simple biologic conception of epithelial activities in the terms of primary, secondary and tertiary hyperplasia rather than to attempt a scientific grouping of cases according to an unscientific and chaotic nomenclature.

REFERENCES

1. Koenig. Lehrbuch der spezielle Chir., 1904, 8 Aufl. ii. Zentralbl. f. Chir., 1893, No. 3.
2. Warren. J. Am. M. Ass., 1905, July 15, 149–165.
3. Bloodgood. Surg., Gynec., & Obst., 1906, iii, 721.
4. Adami and Nicholls. Special Pathology.
5. Kaufmann. Spezielle Pathol. Anat., 1911.
6. Aschoff. Spezieller Teil, Pathologische Anatomie, ii.
7. Borst. Die Lehre von den Geschwülsten.
8. Ziegler. Lehrbuch der Spec. Path. Anat.
9. Forgue. Precis de pathologie extreme (collection Testut).
10. American Textbook of Pathology.
11. Delafield and Prudden. Textbook of Pathology.
12. Mintz. Zentralbl. f. Chir., 1911, No. 6. Berl. klin. Wchnschr., 1899, No. 47.
13. Ribbert. Allgemeine Pathologie, 1901.
14. Lichtenhahn. Ztschr. f. Chir., 1907, xc.
15. Tietze. Deutsche Ztschr. f. Chir., 1900, lvi.
16. Saar. Arch. f. klin. Chir., 1907, lxxxiv, No. 1. Zentralbl. f. klin. Chir., 1907, No. 51.
17. Schimmelbusch. Arch. f. klin. Chir., 1892, xliv.
18. Brissaud. Arch. de Phys. et Path., 1884, iii.
19. Sicre. Thèse de Paris, 1890.
20. Reclus. Rev. de Chir., 1883, No. 10. Pro. Med. 1884, No. 19. Gaz. d. hôp., 1884.
21. Maly. Ztschr. f. Heilkunde, 1898, xix.
22. Roloff. Deutsche Ztschr. f. Chir., 1900, liv.
23. Stiles. Edin. M. J., 1892, xxxvii.
24. Keibel. Berl. klin. Wchnschr., 1904, No. 30.
25. Kuru. Deutsche Ztschr. f. Chir., 1909, xcviii.
26. Bobbio. Giorn. della R. Acad. di Med. di Torino, 1905.
27. Fremicourt. Thèse de Lille, 1909.
28. Brehm. St. Petersb. med. Wchnschr., 1910, xxxv.
29. de Quervain. München. med. Wchnschr., 1908, No. 21. Zentralbl. f. Chir., 1908, 35. Cor.-Bl. f. schweiz., Ärzte, 1910, 40.
30. Verga. Il Policlinico, 1907
31. Franko. La clinica chirurgica, 1906, No. 2.
32. Sasse. Arch. f. klin. Chir., 1897, liv; 1907, lxxxiv.
33. MacCarty. Tr. South Surg. & Gynec. Ass., 1910, xxiii. Collected Papers of the Staff of St. Mary's Hospital, 1911.

LITERATURE ON THE EMBRYOLOGY OF THE SKIN

Backmund. Entwicklung des Haares und Schweissdrüsen des Katzen. Diss., Bern, 1904; and Anatom. Hefte, xxvi, Nos. 2–3.

Chodakowski. Anatomische Untersuchungen über die Hautdrüsen einiger Säugethiere. Dorpat, 1871.

Dien, Franz. Beiträge zur Entwicklung der Schweissdrüsen, etc. Anat. Hefte, xxxiv, 196.

De Sinety. Recchrches sur la mamelle des enfants nouveau-nés. Arch. de physiol., 1875.

Davies. Entwicklung der Feder und ihre Beziehungen zu anderen Integument-abgebilden. Morphol. Jahrb., 1899, xv.

De Meijere. Über die Haare der Säugetiere, besonders über ihre Anordnung. Morphol. Jahrb., 1894, xxi.
De Snoo, D. Over de entwickeling van de epitheliumcel. Inaug.-Diss., Utrecht, 1879.
Eggeling. Über ein wichtiges Stadium in der Entwicklung des menschlichen Milchdrüse. Anat. Anz., 1904, xxiv.
Feiertag. Über die Bildung des Haares. Diss., Dorpat, 1871.
Flatten. Untersuchungen über die Haut des Schweines. Diss., Berlin, 1894.
Ficatier. Études anatomiques des glandes sudoripares. Paris, 1881 (Citiert nach Backmund und Hoffmann, Nr. 2 und 14).
Gurlt. Vergleichende Untersuchungen über die Haut des Menschen und des Haussäugethieres. (Müller's Arch. f. Anat. u. Phys., 1835).
Graff. Vergleichende anatomische Untersuchungen über den Bau der Hautdrüsen des Haussäugethieres und des Menschen. Leipzig, 1879.
Gurlt. Untersuchungen über die hornigen Gebilde des Menschen und des Haussäugethieres. Müller's Arch., 1836.
Götte. Zur Morphologie des Haares. Arch. f. mikr. Anat., 1868, iv.
Greefberg. Die Haut und deren Drüsen in Entwicklung. Mitt. a. d. embryol. Inst., d. k. Univ. in Wien, 1883.
Gegenbaur. Bemerkungen über die Milchdrüsenpapillen der Säugethiere. Ibidem, s. 204.
Gegenbaur. Zur genaueren Kenntniss der Zitzen der Säugethiere. Morphol. Jahrb., 1876, i.
Gegenbaur. Grundriss der vergleichenden Anatomie, 2 Aufl., 1878.
Gurlt. (Handbuch der vergleichenden Anatomie der Haussäugethiere, 4 Aufl.) Sah die Entstehung der Milchdrüsenanlage bei dem Rinde in der 7 Woche; Fürstenberg (Die Milchdrüse der Kuh., Leipzig, 1868). In der 5–8 Woche bei einer Länge des Körpers von 1¾–2 Z.
Huss. Beiträge zur Entwicklungsgeschichte der Milchdrüse. Jenaische Ztschr., 1873, vii, 176.
Hoffman. Über Talg- und Schweissdrüsen. Inaug.-Diss., Tübingen, 1898.
Harms. Beiträge zur Histologie des Hautdrüsen der Haussäugetiere. Hanover, 1868. (Citiert nach Jess, Nr. 15).
Herman, L., and Luchsinger, B. Über die Secretionsströme der Haut bei die Katze. Pflüger's Archiv., 1878, xv, 213.
Heusinger. Beiträge zur Kenntniss der Feder- und Haarbildung. Deutsche Arch. f. d. Physiol. von J. E. Meckel, 1822, vii.
Hertwig. Lehrbuch der Entwicklungsgeschichte, 1888.
Jess. Beiträge zur vergleichenden Anatomie der Haut der Haussäugethiere. Inaug.-Diss., Basel, 1895.
Kushetzoff. Beiträge zur Entwicklungsgeschichte der Kutis. Wien. Sitzungsber. der Kgl. Akad. der Wissensch, 1867.
Kölliker. Lehrbuch der Entwicklungsgeschichte, 1879.
Kölliker. Zur Entwicklungsgeschichte des äusseren Haut. Zeitschr. f. wissensch., h. 1, Zoologie, 1850. iii.
Krause, W. Die Entwicklung der Haut und ihrer Nebenorgane in O. Hertwig's Handbuch der experimentellen und vergleichenden Entwicklungslehre, 1902.
Kölliker. T. Beiträge z. Kenntniss der Brustdrüse. Würzb. Verhandl., 1880, N. F. xiv, 142.
Kölliker. Entwicklungsgeschichte des Menschen und der höheren Thiere, 2 Aufl., s. 773.
Langer. Über den Bau und die Entwicklung der Milchdrüsen. Denkschr. d. Wien. Akad., 1858, iii.
Langer. Die Milchdrüse. Stricker's mikrosk. Anat., s. 627.
Langer. Über den Bau und die Entwicklung der Milchdrüse bei beiden Geschlechtern. Denksch. d. Wien. Akad. d. Wissensch., 1851, iii, Lief. 2.
Luschka. Die Anatomie der Brust des Menschen, 1863, s. 243.
Lüneburg. Beiträge zur Entwicklung und Histologie der Knäueldrüsen in der Achselhöhle des Menschen. Inaug.-Diss., Rostock, 1902.
Leydig. Über die äusseren Bedeckungen der Säugethiere. Müller's Arch. f. Anat. u. Physiol., 1859.
Maurer. Haut-Sinnesorgane, Feder- und Haaranlagen und deren gegenseitige Beziehungen. Morphol. Jahrb., 1892, xviii.
Maurer. Die Epidermis und ihre Abkömmlinge. Leipzig, 1895.
Maurer. Das Integument eines Embryo von Ursus. Arctos. Denkschr. d. med. naturwiss. Ges., Jena, 1904, xi. (Festschr. z. 70 Geburtstage von E. Haeckel.)
Marks. Untersuchungen über die Entwicklung des Hautes, insbesondere des Haars und Drüsenanlagen bei den Haussäugethieren. Diss., Berlin, 1895.
Oyama. Entwicklungsgeschichte des Deckhaares der weissen Maus. Anatom. Hefte von Merkel und Bonnet, 1904.
Okamura. Zur Lehre über die Wachstumsrichtung des Haares in des ersten Anlage. Monatschr. f. prakt. Dermat., 1899, xxviii.
Pasini. Sulla presenza del l' orlo a spazzola nelle ghiandole sudorifere. Monit. zool. ital., Anno 14, No. 5, 1904.
Rein, G. Untersuchungen über die embryonale Entwicklungsgeschichte der Milchdrüse, i.
Rauber. Über die Absonderung der Milch. Schmidt's Jahrb., 1879, 181. 14.
Rauber. Bemerkungen über den feineren Bau der Milchdrüsen. Schmidt's Jahrb., 182, 7.
Reissner. Beiträge zur Kenntniss des Haares des Menschen und des Säugethieres. Breslau, 1854.
Stöhr. Entwicklungsgeschichte des menschlichen Wollhaares. Anat. Hefte, xxiii, 1903.
Stöhr. Entwicklungsgeschichte des menschlichen Wollhaares. Anat. Hefte von Merkel und Bonnet, 1904.
Sticker. Über die Entwicklung und den Bau des Wollhaares beim Schaf. Diss., Berlin, 1887.
Stirling. Beitrag zur Anatomie der Kutis des Hundes. Ber. über d. Verhandl. d. k.-s. Gesellsch. d. Wissensch., 1875. (Ref. von Jess, Nr. 15.)
Talma, S. Beitrag zur Histogenese der weiblichen Brustdrüse.
Unna. Fortschritte der Hautanatomie in den letzten 5 Jahren. Monatschr. f. prakt. Dermat., 1888, vii.
Waldeyer. Atlas des menschlichen und thierischen Haare, sowie ähnlicher Fasergebilde. Lahr., 1884.
Wendt. Über die menschl. Epidermis. Müller's Arch. f. Anat. u. Phys., 1834.
Wilson. Beiträge zur Entwicklung die Haut des Menschen. Mitt. a. d. embryol. Inst., Wien, i, h. 4, 1880.

A SIMPLE METHOD OF EXCLUDING THE PYLORUS

Preliminary Report

By WILLARD BARTLETT, A. M., M. D., Saint Louis, Missouri

A CONSIDERABLE experience in exclusion of the human pylorus, by cutting in two and infolding the coats of the stomach, has convinced me of the desirability of a simpler method of amplifying gastro-enterostomy in certain ulcer cases.

During the past two years I have performed a large number of dog operations, at the Surgical Laboratory of Washington University, with the hope of developing a simple method which should at the same time be clean, safe and permanently effectual.

Every conceivable plan was tried, until by a gradual process of evolution the one here illustrated forced itself upon me. None of the ten dogs on which it was used died from the effects of the operation. The excluded portion was pale and strikingly atrophic at autopsy in each case, while X-ray showed the obstruction to be complete and the gastro-enterostomy patent in the three which were examined months after operation.

In a forthcoming paper will appear all the details of my clinical and laboratory work on pyloric exclusion in gastric and duodenal ulcer.

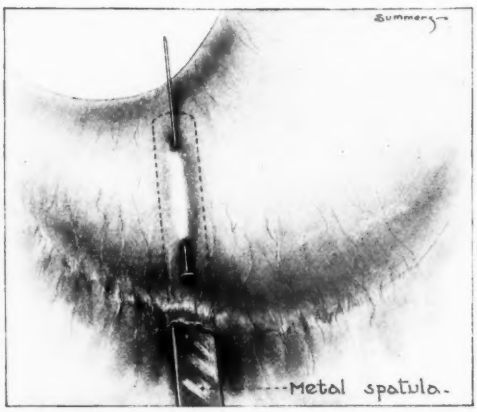
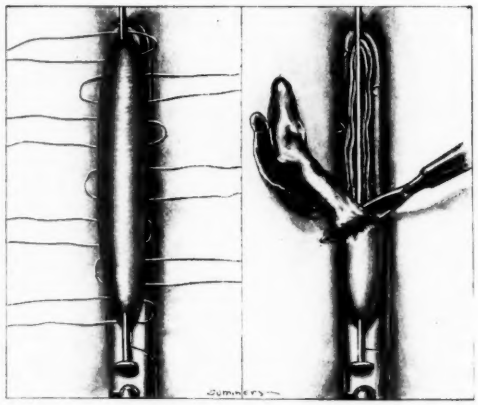
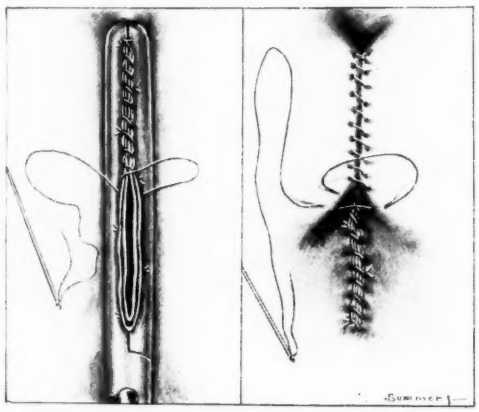


Fig. 1. A metal spatula is introduced through a slit in the gastro-colic ligament to protect the structures behind the stomach, then a metal skewer is thrust directly through both walls of the stomach into the lesser peritoneal cavity and out again so as to encompass about one half the distance between the two curvatures.



Figs. 2 and 3. A clamp is placed behind the skewer and several mattress sutures introduced between the skewer and clamp; as a matter of course these embrace the whole thickness of both walls of the stomach. The skewer is cut out with a sharp knife and the mattress sutures tied before the clamp is removed.



Figs. 4 and 5. A hæmostatic running stitch of silk or linen unites the four exposed cut edges (it may be inserted before the clamp is removed). The ridge of tissue formed by the mattress sutures is inverted with a continuous Lembert row.

INFANTILE TYPE OF UTERUS WITH DYSMENORRHŒA[1]

Report of Cases Treated During the Last Eight Years

By THOMAS J. WATKINS, M. D., Chicago

THERE may be some error in including all of the cases comprised in this report as infantile type of uterus. It is impossible to always accurately classify dysmenorrhœa cases. We have tried to exclude all of the other types of dysmenorrhœa.

There is an important difference between cases of dysmennorrhœa that date from puberty and cases where menstruation was painless during the earlier periods. The first almost invariably occur in the infantile type of uterus. The latter occur from an infection or an impoverished general nervous state. This distinction is important in determining the indications for treatment.

Dysmenorrhœa occurs most often in patients with defective development; in patients where more time has been given to mental than to physical development. Prophylaxis is the most important part of the treatment of dysmenorrhœa. Good hygiene during the first year of menstrual life would minimize the number of cases of dysmenorrhœa. At this time the body generally develops rapidly. This causes a large demand upon the blood-producing organs and anæmia often occurs. A subnormal general physical condition often results in imperfect development of the organs of reproduction.

The girl during the first year of menstrual life should be kept out of school and most of her time should be spent outdoors.

There is probably much to be learned about the relation of ovarian secretion to dysmenorrhœa. It is likely that hypersecretion of the ovary produces an excessive stimulation of the uterus. This may account for the excessive bleeding, clotting and severe contractions in some cases of severe dysmenorrhœa.

The difference in sensitiveness at the internal os in dysmenorrhœa cases is often very marked, and this difference has not been accounted for by pathologic findings. Some cases can barely tolerate the passage of a fine probe through the internal os, while in others considerable dilation can be made without causing much pain.

There is much in common in cases of dysmenorrhœa and the first stage of labor. In both the cervix softens, dilates, and the pains are intermittent.

REPORT OF CASES

This report includes sixty cases of infantile type of uterus with severe dysmenorrhœa treated in the hospital either by laminaria tents or intra-uterine stem pessaries. None of the cases gave a history of, or showed signs of, pelvic infection. The sixty cases so treated represent a relatively small percentage of the patients with painful menstruation seen during this period of time.

In many the dysmenorrhœa seemed to be chiefly a local manifestation of an abnormal general condition. For some, palliative remedies were prescribed, and cervical dilation in the office was occasionally the treatment used.

Use of tents. Tent dilatation was employed in the earlier cases. The patients remained in the hospital one to three weeks. One or more small tents were used in preference to larger ones. They were used repeatedly, with two or three day intervals when necessary, to obtain free dilatation. In an occasional case tissue which was difficult to thoroughly dilate was encountered at the internal os. The tents were usually left in forty-eight hours, as earlier removal was found difficult because of a constricting ring of tissue at the internal os. Even at the end of forty-eight hours it was occasionally difficult to remove the tent. This occurred less often after use of smaller sized tents was instituted. The firm band of tissue at the internal os was in a few cases incised anteriorly.

After a number of patients had been

[1] Read before the Chicago Gynecological Society, April 18, 1913. See discussion p. 516.

treated by the use of tents they were discontinued, as one patient developed an ovarian abscess and another a pelvic exudate.

Use of intra-uterine stem pessary. The intra-uterine stem pessary was used in cases subsequently treated. It is used with the intention that it produces hypertrophy such as results when a fibroid, fœtus, or foreign body is present in the uterus.

Technique. The same aseptic care is used as in the preparation for any vaginal operation. Careless surgery might produce much harm, as the intra-uterine stem is to be left in place for some time.

An anæsthetic is used, preferably nitrous oxide gas, as free dilation facilitates introduction of the stem and diminishes the after-pain.

Considerable experimental work was done to find a stem that would remain in place. Some were made with a large distal end, but were found to be unsatisfactory. We have been unable to keep any of them in without suture, and in a relatively short time the suture generally cuts through the tissue.

Dr. R. L. Dickinson informs me that he fastens the stem with a large silver wire suture and finds that it does not cut through the cervical tissue.

We are now using the stem here shown. (Fig. 1.) A suture is passed through either lip of the cervix, otherwise one lip may stretch and allow it to be forced down through the internal os.

The patient is kept in the hospital until all pain has subsided. This time has varied in different patients from a few hours to one week. The instrument is allowed to remain for four to six weeks, if it is not spontaneously expelled earlier.

Results. We have endeavored to ascertain the present condition of these patients. Letters were sent to all whose present condition was not known. The following results were obtained:

Cured, 15 (25 per cent). This does not mean that all of the patients were relieved of all pain, but that they ceased to be confined to bed or to take anodynes and were satisfied with the result. Improved, 10 (16+ per cent). No improvement, 6 (10 per cent). Failed to reply, 15. Letters returned, 14.

The following cases present features of interest:

Miss G. was dilated with tents and later had a stem inserted. No relief resulted. An effort was made to dilate at my office before each menstruation. This was unsatisfactory on account of the pain which resulted. A supra-vaginal hysterectomy was finally made. For six months after operation was performed she continued to menstruate scantily from the cervix, and stated that she had excruciating pains. We have been unable to ascertain her condition during the last year.

Miss S., a school teacher, complained of severe dysmenorrhœa which confined her to bed for two or three days each month. Examination showed no pathologic condition. The following summer was passed in Europe. While away the menstrual periods were painless. On resuming teaching the following year, the dysmenorrhœa recurred. Her improved general health while abroad was the probable cause of the painless menstruation.

A study of the sixty cases emphasizes the fact that much remains to be learned about dysmenorrhœa cases, that the treatment is far from satisfactory.

SUMMARY

1. Dysmenorrhœa is most often due to imperfect development of the uterus.

2. The pain is chiefly caused by contractions of the uterus, which soften and dilate the cervix and are much like the pains of the first stage of labor.

3. Prophylaxis is the most important part of the treatment. The general health of the girl should be carefully looked after during puberty.

4. Dysmenorrhœa is often due in part or entirely to the general condition of the patient. In cases of infantile uterus causing dysmenorrhœa the logical treatment consists in the use of remedies to promote development. The use of the intra-uterine stem is useful in such cases. The patient should be kept under supervision for months after the use of the stem pessary. The cervix should be dilated when possible before each period for some months. The stem pessary should

be reinserted after one or two months if necessary. This treatment is indicated only in the severe type of cases. It is, however, a poor substitute for marriage and pregnancy.

In cases of excessive menstruation with clots and severe pain relief may be obtained from the use of suprarenal gland, which has lately been recommended. Suprarenal gland diminishes the action of the ovarian secretion. One three-grain tablet of the desiccated gland is given three or four times daily, for three days just before or during the early part of the menstrual period. We have not used this sufficiently to be prepared to report upon its value.

5. There is much to be desired relative to our knowledge and treatment of this type of cases.

MALIGNANT HYPERNEPHROMA OF THE KIDNEY, ITS CLINICAL COURSE AND DIAGNOSIS, WITH A DESCRIPTION OF THE AUTHOR'S METHOD OF RADICAL OPERATIVE CURE[1]

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IN THE Mount Sinai Hospital reports for 1905–6, Vol. v., the writer published the cases of malignant hypernephroma that had been admitted to all the services of the hospital from 1898 to January, 1907. During this period there were admitted to the hospital twenty-five cases of renal neoplasm, twenty-one of which proved to be hypernephroma, or perithelial sarcoma of the kidney; that is, 87½ per cent of the cases of renal tumor belonged to the class of hypernephroma. This corresponded roughly with the frequency of occurrence of this malady in the Allgemeiner Krankenhaus in Vienna From this clinic, Albrecht reported twenty-eight cases of hypernephroma out of thirty-two cases of renal tumor. Block, out of one hundred and twenty-six cases of malignant tumor at the Israel clinic, reported eighty-six as being hypernephroma.

Since January, 1907, there have been on the first surgical service of Mount Sinai Hospital thirteen additional cases of renal neoplasm, of which nine were undoubtedly hypernephroma; two more were clinically hypernephroma, but inasmuch as they were not operated upon this clinical diagnosis could not be substantiated by an histological one; one was a mixed tumor — myxosarcoma and adenocarcinoma, and one was a carcinoma secondary to renal calculi.

In this new series of thirteen cases, 70 per cent were histologically hypernephroma, 84 per cent plus were clinically hypernephroma, which is almost the same proportion, as in our previous report. From these figures and from those of Albrecht and Block we must infer that hypernephroma is by far the most common of renal tumors.

In our new series seven of the undoubted cases of hypernephroma were in adults, varying from twenty-two to sixty years of age; one was in a child of six years of age.

The term hypernephroma was applied by Birch-Hirschfeld to tumors arising from adrenal tissue located elsewhere than in the suprarenal gland. Such islands of adrenal tissue, called adrenal rests, occur most frequently in the kidney beneath its capsule. They have also been found under the capsule of the liver along the course of the spermatic artery, in the spermatic cord, and on the peritoneum near the ovaries. Tumors arising from such adrenal rests have been designated as hypernephromata. Wilson and Ribbert have recently maintained that hypernephromata originate from the remains of the Wolffian

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bodies. These two authors deny the origin from adrenal rests.

It is the intent of the author in this paper to consider the relative malignancy of hypernephromata, its diagnosis, and especially the method of removal of the tumor.

The question of the relative malignancy of hypernephroma is a disputed one. Writers upon this subject differ very considerably on this point. Thus Block, writing from Israel's clinic, reports about one-third of the cases as being radically cured by operation, whereas Krönlein could only report two cases alive out of eleven, one, one and a half years after operation, and one six years after the operation. Of our own cases all those in the first series have died, and of the present series six have died, two are living eight and nine months, respectively, after the operation, and one has been operated upon very recently.

Our own statistics, therefore, bear out the view of Krönlein that hypernephroma is a very malignant, if not the most malignant of renal tumors. On the other hand, it should be distinctly noted that hypernephroma can, and sometimes does, follow a very benign course.

Thus a patient of the writer's has had a hypernephroma of the left kidney for at least ten years. During this time she has suffered with repeated attacks of hæmaturia, and some five years ago the writer operated upon her for an acute gangrenous cholecystitis, from which she made a smooth, uninterrupted recovery. The hypernephromatous tumor has not been removed on account of her poor physical condition and yet during these ten years she has not lost ground materially. She is now over sixty years of age, attends to all of her work, and feels well. Of course, it is not positive that her left renal tumor is of a hypernephromatous character, yet it is a nodular, irregular mass, with areas of hardness and softness, it bleeds from time to time, and in every respect resembles our other cases of hypernephroma.

Another one of our patients was admitted to the hospital for intense sciatic pains. The man was in excellent physical condition and had been treated for months with all the remedies that are usually employed for the treatment of sciatica. On rectal examination a pulsating tumor was felt in the lower portion of the pelvis, close to the lateral pelvic wall, and adherent to the ischium. This pulsating tumor was thought to be an aneurism of the gluteal artery, and for its cure ligation of the internal iliac artery was practiced. The patient succumbed from peritonitis, and on autopsy the tumor was found to be a hypernephroma, secondary to a small hypernephromatous tumor in the kidney. The patient was not cachectic, gave no evidence at all of the primary hypernephroma in his kidney.

How shall such radically different clinical courses of this malady be explained? On the one hand that of a most malignant tumor and on the other that of a benign affection.

It is readily conceivable that hypernephromata may at one period of their course be of a benign character, growing very slowly, giving few or no symptoms (occasional hæmaturia, rarely a palpable tumor), not interfering with general well-being and differing from other benign tumors only in the fact that they may occasion metastases in distant organs. Pathologists may claim that benign tumors never give rise to metastatic deposits, but hypernephromata like adenomata of the thyroid, which histologically they strongly resemble may cause metastases and yet be entirely benign in their clinical course. The explanation of this is seen when we look at the minute anatomical structure of hypernephroma. The simplest types of these tumors consist of alternating columns of cells and blood vessels, and because of this intimate relation between the cells and blood vessels, it is possible for fragments of the cells to be dislodged into the blood stream and carried to other organs. Thus there have been reported in the literature several cases of secondary hypernephromatous tumors in the bones, etc., with no evidence of a primary kidney tumor, and no clinical manifestations of malignant disease, and with a long period of life after the diagnosis of metastatic hypernephroma was made. Such metastases are usually single but they may be multiple, and when they are removed they are not likely to recur. We have no knowledge of how long this benign period lasts. It may be for many years. As most of the patients with malignant hypernephroma are of full adult life, and as the neoplasm arises from fœtal remains it is more than likely that the tumor formation occurred therein at a much earlier period of life, progressed slowly and gave no marked symptoms. Hence it remained unperceived and unrecognized, unless a metastasis formed in

a superficial organ and so permitted of the diagnosis being made. At times the primary renal tumor grows to large size, as in the woman whose history has been cited above. The occasional occurrence of malignant hypernephroma in children would bear out the view that early tumor formation in the fœtal remains (whether of adrenal rests or Wolffian bodies) does occur.

At some time or other a malignant degeneration of the benign hypernephroma takes place. Then commences the active period of the disease, and from now on the tumor manifests all the signs of extreme malignancy. It grows rapidly, infiltrates neighboring structures, bleeds profusely, causes marked cachexia and death.

To sum up the remarks just made:

Hypernephromata commence to develop early in life from fœtal remains of adrenal rests or Wolffian bodies; at the earliest period of their development they are benign tumors, usually of small size, occasionally of quite marked proportions, giving no symptoms of their presence, unless by their size or by a metastasis in a superficial organ. At some time, maybe in childhood or in adult life, a malignant degeneration of the benign tumor takes place, and then commences the active period of the disease in which the patients come under our observation. At this time the tumor is of the greatest malignancy. Only in such a way can we reconcile the diverse clinical manifestations of hypernephromata and the different views of clinicians as to the relative malignancy of this type of neoplasm.

Now as to the symptoms and diagnosis of hypernephromata. As was just said, during the benign period it is rarely possible to recognize the primary focus of the disease. The tumor is small, it usually does not bleed, and it does not interfere with the general well-being of the individual. Now and then the tumor will grow to considerable size and then be recognized; again, a metastasis in the bones or some other organ will occasion symptoms that lead to the recognition of a tumor and the histological examination thereof will reveal its character. But in the active or malignant period of the disease the symptoms develop rapidly. Most of the patients suffer only for a short time before they seek medical advice. In our present series the longest period during which symptoms were manifested was two years, and the shortest a few months.

In our first series the largest percentage of patients, six of the twenty-one, presented as an initial symptom a dull ache or sharp pain in the loin or abdomen, which sometimes radiated to the lower extremities. In the present series this pain was noticed in six of the eleven patients. It was not of a colicky character. This pain is variously explained; by some it is believed to be due to pressure on the lumbar plexus, by others to increased kidney tension occasioned by hæmorrhages within the tumor. This pain may exist some time before any other symptom or physical sign appears. Albrecht and Israel consider that a long duration of lumbar pain prior to the occurrence of hæmaturia is of diagnostic importance in the differentiation of hypernephroma from other varieties of renal neoplasm. In some of these patients there will be microscopic blood in the urine for some time prior to the occurrence of macroscopic hæmaturia: therefore the importance of a careful urinary analysis for red blood cells in all cases of persistent lumbar pain, of which the causation is in doubt, is very evident.

In one of our present series the initial symptom was a sudden onset of pain in the lower portion of the right chest.

The patient thought the pain was due to his having slept by an open window, and reported that it was made worse by breathing. There was no cough, no fever, occasional sweats, and the right lower thoracic region was tender to pressure. This pain and tenderness lasted four weeks, and then improved so that the patient could return to work. Three weeks later he was struck in the right scapular region by a roll of oilcloth, and following this a swelling the size of a walnut appeared at the site of the injury. This swelling gradually increased in size, — it was punctured several times by his family physician, and only blood was obtained. He lost flesh and strength, and was markedly constipated. His urine contained red blood cells. On his admission to the hospital there was an irregularly oval, smooth, semi-fluctuating tumor, extending from the spine to the right posterior axillary line over the seventh, eighth and ninth ribs. There was no tumor

palpable in either loin, or in the abdomen, though when pressure was made in the right upper quadrant of the abdomen, pain referred to the site of the tumor was complained of. The liver outlines were approximately normal to percussion. There was slight dullness from just below the angle of scapula to the base on the right side, and over the dull area the breath sounds were slightly diminished. At operation, the very vascular, soft and necrotic tumor was found to extend into the thorax, below the level of the diaphragm, which had been pushed upward by it, and into the right kidney region; posteriorly it had invaded the bodies of the lower dorsal vertebræ.

In discussing this tumor with the pathologist, Dr. Mandlebaum, prior to its histological examination, he suggested hypernephroma, and chiefly because of the presence of red blood cells in the urine. Persistent lumbar pain then without assignable cause, and red blood cells in the urine, are strongly suggestive of hypernephroma.

Hæmaturia, microscopic or macroscopic in character, occurs in a number of the patients with hypernephroma. The importance of examining the urine for microscopic blood has already been dwelt upon.

Macroscopic hæmaturia is in many cases the earliest symptom of the disease (in one case of present series). The hæmaturia is usually attended with colicky pain radiating to the bladder, frequent urination and tenesmus. Sometimes the bleeding is profuse, again slight, and sometimes there are long ureteral casts of blood in the urine. In comparison with other malignant kidney tumors the hypernephromata are less likely to be attended by hæmaturia. Thus Israel computed that 92 per cent of the cases of malignant kidney neoplasm are attended with macro- or microscopic hæmaturia, and that in 70 per cent this is the initial symptom. Of Albrecht's hypernephroma cases, 39.3 per cent had blood in the urine at some time of the disease, whereas in only 21.4 per cent was it the initial symptom.

In our first series hæmaturia was the initial symptom in 13.6 per cent, while in 40 per cent there was blood in the urine at some time or other. In our last series, blood was present in the urine in four; i. e., 36 per cent (macroscopic in three, and microscopic in one), and no blood in the other seven. A study of the specimens of hypernephromatous kidney will explain why hæmaturia is absent in so many instances. In quite a number the entire secreting structure of the organ has been destroyed, and the pelvis thereof completely filled with tumor masses. As such organs secrete no urine, there is no product to contaminate the healthy urine of the other kidney, and as the pelvis is choked with tumor masses no blood can make its way through it into the ureter and bladder. In two of the first series, and in three of the present series of cases, such a condition existed: In some patients the hæmaturia is the first symptom to attract the patient's notice, but examination will reveal a more or less large tumor in the loin; but in others no physical abnormality can be detected by palpation in either loin. This latter does not mean that no tumor is present, for a tumor at the upper pole of the kidney, by displacing the diaphragm upward may attain even large size, and yet not be palpable in the loin. Israel believes that a long interval between the attacks of hæmaturia points to the probable diagnosis of hypernephroma.

Progressive loss of weight, weakness and anæmia are often the earliest symptoms of hypernephroma. This was the case in three patients of the first and in one of the second series. In these patients there is usually a large, rapidly growing tumor present. Sometimes the presence of a tumor in the abdomen is the first abnormal thing that is noticed, and in a majority of the patients a tumor becomes palpable in the loin sooner or later in the disease.

In our first series 51 per cent had a palpable tumor in the loin at the time of their admission to the hospital, and in our present series nine out of eleven had such a palpable mass. The tumor varied in shape, size, consistency and fixidity. In some instances it was rather small, hard and nodular; the larger tumors were of unequal consistency, hard in places, softer and elastic in others, and semi-fluctuant. The nodular character seemed to belong to all the tumors, and Israel considers this of differential diagnostic value from other malignant kidney tumors. The smaller tumors are usually freely movable; the larger ones are more or less firmly fixed.

As has already been stated, the presence of one or more metastatic tumors is sometimes the very first evidence of the disease, and depending upon whether the metastasis occurs in the benign or malign period of the disease will the secondary deposit manifest the characteristics of a kind, slowly growing tumor or of a rapidly growing, ulcerating malignant neoplasm. In two of our first series of cases such metastases (one in the ischium and one in the frontal bone) were the first evidences of the malady, and in both patients the secondary tumors acted in a benign way. One patient succumbed to peritonitis from transperitoneal ligation of the internal iliac artery, done because the tumor was supposed to be an aneurism of the internal iliac artery, and the other patient remained well for several years after the removal of the frontal bone tumor, but ultimately succumbed.

The metastases are most frequently located in the long bones, in the bones of the skull, sternum or pelvis, or in the lung. They are usually painful and when situated in the bones they do not excite any periostitis, but gradually cause erosion of the cortical substance, and so occasion spontaneous fracture. This latter fact is of importance in the X-ray diagnosis of such bone tumors. Though the metastatic tumor is often the first symptom *noticed*, yet frequently a physical examination will reveal a nodular tumor in the loin, or a careful urinary examination will show the presence therein of red blood cells. Hence the importance of making a thorough physical examination and urinary analysis in all cases of neoplasms, and most especially those of bones. This was well illustrated in the patient with a thoracic tumor whose history was cited above; here the presence of red blood cells in the urine led to the diagnosis of hypernephroma.

It is to be noted that the metastases of hypernephroma are very often single; this has an important bearing in the consideration of the treatment of such cases; for whereas with other varieties of malignant tumors we would decline to remove either the primary or secondary tumor, because the metastatic foci of the disease are apt to be multiple, yet in this variety of growth we would be justified in extirpating both the primary tumor and the secondary deposit.

The metastases from hypernephromata are especially likely to take place through the blood stream, though the writer has recently found the neighboring lymphatic glands and lymphatics enlarged in several cases. The reason of this tendency to spread through the medium of the blood stream lies in the fact that tumor cells bear a most close and intimate relation to the blood vessels, and also because these neoplasms frequently extend and grow into the renal vein and inferior vena cava. Israel found a thrombosis of the renal vein with tumor masses in thirteen of his eighty-six cases, and the writer found it in several of his cases.

In one recent case the renal vein and inferior vena cava were filled with tumor masses, most of which were removed at the time of operation. This spread of the tumor to the large blood vessels is especially important when we consider the technical details of the removal of these tumors. In several of our patients there developed after operation symptoms of general systemic intoxication, which seemed to result from the dissemination of tumor tissue by the manipulations connected with its removal. The fear of such dissemination, and the attempt to do a complete radical cure, had led the writer to his present method of operating.

Israel has laid considerable stress upon moderate temperature elevations occurring with hypernephroma; we have noticed such temperature elevations whenever a large tumor was present, especially when the latter was rapidly growing, and have ascribed this either to hæmorrhage into the tumor or to the absorption therefrom into the circulation of necrobiotic matter.

In a detailed discussion of the diagnosis and differential diagnosis of hypernephroma it is well to consider the disease in its initial and later stages. First, as to those cases in which pain in the loin is the first symptom. Some of the patients will endure the pain for quite a time before they seek medical advice, and when they finally do consult the physician there may be a palpable, nodular tumor in the loin. In such cases a long duration of the pain

is suggestive of the tumor being a hypernephroma. Other patients endure the pain until an attack of hæmaturia occurs; in such cases a long interval of pain prior to the occurrence of hæmaturia suggests the diagnosis of hypernephroma. Altogether in hypernephroma, lumbar pain is more apt to be a striking symptom than it is with the other varieties of renal neoplasm.

Should the patients of this class seek relief in the early stages of their suffering, i. e., prior to the appearance of a tumor or to the occurrence of hæmaturia, then the diagnosis cannot be arrived at. In such cases we will have to consider all the possible causes of pain in the loin, and by thorough examination and careful observation try to exclude first one and then the other cause. In these patients it is especially important to examine the urine repeatedly for microscopic blood cells. If red blood cells are present, the finding will be especially significant if it is proved by ureteral catheterization that they come from the kidney corresponding to the side of the pain, and if by X-ray examination we can exclude the presence of renal stone.

It has been pointed out by Israel that where, because of pain in the loin and hæmaturia (whether macro- or microscopic), we suspect the presence of a neoplasm of the kidney, the occurrence of moderate, irregular temperature elevations is suggestive of the tumor being of adrenal type. Thus he has found that in 57 per cent of his cases of adrenal tumors there were these irregular temperature elevations, whereas in only one or two per cent of the other cases of renal tumor did such fever occur. In 10 of our patients, the temperature prior to operation ranged between 99-100°, and in only two did it reach 102°. We have not found in these early stages of this disease that the patients lose markedly in flesh or strength.

The presence of a palpable tumor in the loin makes the diagnosis in these cases somewhat easier. We have first of all to recognize the tumor as a retroperitoneal one, thereby excluding the possibility of its being gallbladder, liver or stomach; secondly, we must differentiate between the varieties of retroperitoneal lumbar tumors, e. g., lymphosarcoma, and other types of kidney tumors, as tuberculosis and pyonephrosis. These diagnostic data can be ascertained by careful consideration of the clinical history, by distention of the stomach and colon, by gastric analysis, by cystoscopic examination, and by analysis of the combined and separate urines.

In this way we can narrow down the diagnosis to some form of renal neoplasm, and in our conclusion as to what type of tumor is present we are guided (1) by the comparative frequency of occurrence of the different varieties of kidney tumor; (2) by the duration of the symptoms; (3) by the physical characteristics of the tumor and its relation to the kidney; (4) by the occurrence of irregular temperature elevations; (5) by the less frequency of hæmaturia with hypernephroma.

It has already been pointed out that hypernephromata are much more frequent than are renal sarcoma or carcinoma; in our series, and in those of Albrecht 87 per cent of all the cases of renal neoplasm belonged to the type of hypernephroma.

Albrecht has also drawn attention to the fact, and this is confirmed by our own observation, that a long interval between the initial symptoms, pain, hæmaturia, etc., and the time the patient applies for treatment points to hypernephroma as against sarcoma or carcinoma. Again, as has been pointed out above, hypernephromata are attended with but little cachexia, whereas in sarcoma and carcinoma this is an early manifestation.

The relation of the tumor to the kidney and its shape will sometimes help us materially in the differentiation. Thus it is a well-observed fact that sarcoma and carcinoma of the kidney cause a uniform enlargement of this organ and therefore the tumor maintains an ovoidal or reniform shape, whereas hypernephroma causes an enlargement only of part of the kidney. The latter tumors, therefore, have an irregular, broad shape, and if the upper pole or lateral aspect of the kidney is involved it may be possible to palpate the normal lower pole of the organ.

The recognition of the disease in those patients whose initial symptom is hæmaturia is often very difficult. Should there be a characteristic palpable tumor in the loin,

much aid will be afforded in making the diagnosis. In the absence of such a tumor, we must first determine by the cystoscope and ureteral catheter from which kidney the blood is derived; then by X-ray examination, physical examination and urinary analysis, exclude calculous disease, tuberculosis and chronic inflammation, and finally resort to diagnostic incision when any doubt exists as to the nature of the kidney malady.

As was stated before, it is our opinion that in all those patients whose primary and chief complaints are loss of weight and strength, increasing weakness and lassitude, there is a rapidly growing and palpable tumor in the loin or metastatic tumors in other organs. The tumor may not have attracted the patient's attention, and he may not seek medical aid until hæmaturia occurs, or until an increase in the size of the abdomen, or a continuous pain in the loin, or the accidental palpation of a lump in the abdomen impels him to apply to the physician. In these patients the characteristics of the tumor will materially aid in reaching the correct diagnosis.

The disease is easiest of diagnosis in patients who come to us complaining only of a tumor in the loin. The relations and characteristics of the tumor, the relative infrequency of macroscopic hæmaturia and the greater frequency of occurrence of hypernephroma, and the exclusion of stone and tuberculosis by X-ray examination, cystoscopy, and ureteral catheterization render the diagnosis easy.

The most interesting cases for diagnosis are those in which the initial manifestation of the disease is a metastatic tumor in the bone or other viscera. So frequently do these metastatic tumors occur in connection with hypernephroma, that it should be our invariable practice, when we have to do with a growth of doubtful character in a bone or internal organ, to carefully palpate the loin for the presence of a tumor and examine the urine for red blood-cells. If we only will remember that tumors of the bones, lungs, or other internal organs may be metastatic to hypernephroma of the kidney and always follow the practice indicated above, many cases will be diagnosed before they reach the post-mortem room and pathological laboratory. It should be noted, however, that there occur cases of hypernephroma with metastatic deposits in which the most painstaking examination will fail to show any abnormality in the urine or in the kidney regions. In our series of patients two were of this kind; but in a third, who came with a painful tumor of the thorax at the angle of the scapula, and in whom long and careful palpation of the loin failed to reveal a kidney tumor, the diagnosis was suggested by the presence of red blood-cells in the urine.

Albrecht thinks that a hypernephroma of the bone can be differentiated from tuberculous osteomyelitis by the absence of periosteal thickening and rarefication, and from medullary sarcoma of bone by the fact that the latter tumors thin out, but do not destroy the cortical tissue until the final stages, whereas hypernephromata destroy all the component layers, viz., the periosteum, cortex and medulla. Such differences are of course only to be appreciated by X-ray examination.

The diagnosis in such cases of this disease as run an atypical course, as the two cases of our series already detailed, cannot be made except by histological and post-mortem examination.

In summing up the remarks just made as to diagnosis, the writer would insist on the importance of the characteristics of the renal tumor, on the much slower course of hypernephroma, on their much greater frequency of occurrence as compared with other varieties of renal neoplasms, on their less frequent association with hæmaturia than is the case with sarcoma or carcinoma of the kidney, and on their greater likelihood to be attended by continued pain in the loin or abdomen. Furthermore, the writer would again insist on the importance of a careful palpation of the kidney regions and urinary analysis in all cases of tumor of the bones, lungs or other internal organs.

Before considering the indications for operation, and the manner of removing the neoplasm, the writer desires again to draw attention to two important points in the pathology.

First and foremost is the intimate relation that exists between the tumor cells and the blood vessels: This accounts for the frequent extension of the tumor into the veins. That such extension may take place while the tumor is clinically benign, and so give rise to a clinically benign metastasis in a distant organ, has already been referred to. Naturally the invasion of the veins is more likely to take place when the tumor has assumed malignant characteristics.

Block reports extension into the renal vein in thirteen of his eighty-six cases; the writer is unable to state the percentage of cases in his series that showed extension of the neoplasm into the renal vein, but he is of the opinion that this is a very frequent complication, and explains why so few cases of lasting cure are effected by the present method of radical cure. Thus in one of the last three cases operated by the writer the vena cava and renal vein were filled with masses of tumor tissue.

This intimate relation of the tumor to the blood vessels is of further importance to the surgeon. The manipulations necessary for the removal of the neoplasm are very likely to inject the tumor tissue into the veins, and so occasion general systemic infection with hypernephromatous material.

It is further important to remember that the metastases in hypernephroma are not infrequently solitary ones. Consequently if such a metastasis together with the primary tumor are widely removed permanent and radical cure is possible. In this respect the hypernephromata differ from sarcoma and carcinoma, for with both these types of tumor the metastases are usually multiple. Thus in one of the cases in our first series there was a solitary metastasis in the ischium, and Albrecht reports a case of single metastasis in the clavicle. Other cases with a solitary metastasis have been reported in the literature. In this respect the hypernephromata resemble the fœtal adenoma of the thyroid gland.

Now as to the indications for operation:

Impressed with the high mortality that immediately attended or shortly followed the removal of large hypernephromatous tumors of the kidney, the writer several years ago took the position that where one had to deal with a large tumor that did not materially interfere with the general well-being of the affected individual, and especially if the patient was an elderly one, or one with diseased or compromised internal organs, it would be better not to interfere surgically.

The bad operative results were partly due to profuse hæmorrhage and shock, and partly to the dissemination of hypernephromatous material into the bloodstream with symptoms of general systemic intoxication (high temperature, rapid pulse, etc). The route that had been followed for the removal of these tumors had either been the lumbar or the anterior transperitoneal one.

But since the plan of operation which will be here described has been practiced, the writer has failed to encounter the previously noted hæmorrhage or shock, or evidences of dissemination of tumor tissue, although very large neoplasms have been removed. Therefore he is more hopeful for the radical cure by operation of even those patients with large hypernephromata, provided the tumor has not infiltrated the surrounding muscular structures.

I would at the present time consider favorable for operation a patient whose general condition warrants surgical interference (this, of course, includes a healthy opposite kidney), and when the local relations of the tumor give us a fair hope of removing the entire disease. A metastatic deposit would not deter me from radical operation, provided careful observation showed only a single secondary growth to be present, for, as has been several times noted, metastases from this tumor are often solitary ones. Of course, it would be advisable in the presence of metastatic deposits to operate upon the primary malady, and the secondary deposits at different sittings.

As to the method to be pursued in the removal of the kidney tumor it is essential that the patient be carefully prepared, and especially that the intestines be thoroughly emptied. The one great aim is to prevent hæmorrhage and dissemination of tumor material into the blood stream. This is accomplished by a free exposure of the aorta,

vena cava, and corresponding renal vein and artery before any manipulation with the renal tumor is made, and in the following way: The patient is placed upon the healthy side with a firm round pillow or table rest under this flank. An incision is then made from the outer border of the quadratus lumborum muscle on the diseased side transversely across the body to the median line in front, the anterior end curving up or down, depending on whether the tumor occupies the hypochondrium or iliac region. The level of the incision is just below the twelfth rib. It is deepened down through all the muscles to the kidney capsule and peritoneum. At the anterior end of the incision only the rectus sheath is incised, the latter muscle then being retracted inward. After careful hemastasis the peritoneal cavity is opened, and the viscera protected by hot compresses, which latter also push and hold the intestines aside. An exploration is now made of the liver and mesentery for evidences of metastases; if none are found the radical operation is proceeded with. The posterior parietal peritoneum just outside of the colon is incised parallel to the course of the colon and the latter together with its peritoneal covering and its mesenteric vessels are stripped up bluntly from the front inner aspects of the tumor, until the aorta, vena cava, and corresponding renal vessels are fully exposed. The cava and renal vein are examined to determine whether there is tumor tissue within them. If so, this is left to be dealt with at a subsequent stage of the operation, the renal vein being now provisionally doubly tied. The renal artery is similarly doubly deligated, likewise the ureter, and then the tumor is rapidly removed in retrograde fashion. It is surprising to see how scanty is the hæmorrhage and how minimal the shock. What little blood is lost comes from a few small vessels that run into the capsule from the surrounding parts, and this is rapidly checked by hot tampons. If the renal vein and cava are free of tumor tissue, drainage is made posteriorly, and the peritoneum and outer wound rapidly closed. If the renal vein and cava contain tumor thrombi, as in one of the writer's recent cases, then the cava should be clamped above and below the thrombus by a serafine forceps, the provisional ligature removed from the renal vein, and the thrombi milked out of the cava and renal vein. If necessary an opening should be made in the cava to facilitate the removal of the tumor mass. After thorough evacuation of the tumor from these vessels the vein should be tied securely, or if the cava has been opened this should be closed by suture — drainage posteriorly and closure of the peritoneum and outer wound as above.

The writer has done this operation six times, with one operative death.

With this method of removing the primary tumor, and dealing with the deposits in the veins, the writer hopes at a future time to report more favorable results from the radical operative treatment of hypernephroma.

BLOOD PRESSURE DURING PREGNANCY[1]

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THE study of blood pressure begins with the work of Stephen Hales in 1733, one hundred and seventeen years after Harvey's first description of the circulation. Poiseuille, in 1828, made the first great improvement in the method with the description of the mercurial manometer, which Ludwig converted into a self-registering instrument, nineteen years later. These methods study blood pressure by direct measurement, and demand in consequence the opening of blood vessels for the insertion of the connecting cannula. Quite naturally, therefore, they are not suitable for clinical determinations on man.

From the myriad of men who have concerned themselves with the development of accurate clinical methods, three names stand out most prominently: Vierordt, who suggested in 1855 the principle of the indirect estimation of blood pressure by the obliteration of the pulse by the direct application of weights to the radial artery; Marey, who in 1876 utilized water under pressure as such weight, while depicting the pulse curve by a recording lever and tambour; and Basch, who in the same year specified the pressure in terms of mercury.

At the present time there are many instruments suitable for clinical use. For the most part they consist of a flat rubber bag encased in an unyielding cuff of 12 cm. width, which is placed about the upper arm and is blown up by air pressure. This is connected with a mercury manometer and dial, or a recorder which is influenced by the expansion of air in a chamber between two thin metal discs, giving its readings in terms of millimetres of mercury.

Two pressures may be taken, and from these others may be calculated. The maximum or systolic blood pressure is that pressure which obtains at the crest of the pulse wave, and may be determined by noting the pressure that is necessary to block the passage of the pulse wave. Taken in the brachial artery it tends to approximate the lateral pressure in the aorta. The minimum or diastolic pressure is that pressure which exists at the trough of the pulse wave; it measures the peripheral resistance, and represents the resistance made by the arteries to the expulsion of blood from the heart. The difference between the systolic and diastolic pressures is called the pulse pressure, and supplies an index of the output of the heart per systole. The greater the difference between the first two pressures, the greater will be the amount of blood entering the arteries. The mean pressure in the arteries may be computed as suggested by Dawson, by adding one third the pulse pressure to the diastolic pressure. Systolic pressure usually has been measured by determining the pressure at which the radial pulse becomes perceptible to the palpating finger after the pressure obliterating the brachial has been gradually released.

Since the auscultatory method was introduced by Korotkow in 1905, it has been shown that the palpatory method gives lower and more inaccurate readings than the former. Thus Korotkow found his determinations 10 to 12 mm. higher with the auscultatory method; Gittmas, differences of an average of 16.7 mm. in 61 cases; while Warfield in a recent paper records variations of 8 to 14 mm.

The systolic pressure should be determined with a small bell stethoscope placed over the brachial artery and just below the compressing cuff. It will be found to correspond to the first sound which appears in the vessel as compression is released sufficiently to allow the blood to rush again into the lower artery.

The phases of tone, described by Korotkow, which are heard during the different stages of pressure upon the brachial artery in man have been modified into five stages by Ettinger, Fischer, and others. The first phase is a more or less sharp, clear tone corresponding to the passage of the first pulse waves

[1] Read before the Chicago Medical Society, March 19th, 1913.

under the compressing cuff. This tone persists during the second phase but is modified by a series of murmurs. The third phase is a transition to a loud, sharp, clear tone, which gradually or suddenly becomes dull at the beginning of the fourth phase. The fifth phase marks the cessation of all sounds. Diastolic pressure has been read frequently in this country at the point of disappearance of all sound, following the statements of Ettinger, Hirschfelder, Gittings, and Goodman and Howell. Korotkow, however, from theoretical considerations placed the diastolic pressure at the point where the clear, loud tone was replaced by a dull tone — the beginning of the fourth phase. This has been confirmed by many observers, among whom we may mention Fischer, Lang and Manswetona, Warfield, and others.

The majority of pressure observations in obstetric literature concerns systolic pressure alone, determined by palpatory methods. Such observations, even though of limited value, possibly, in a comparison with more complete study, have been helpful occasionally in certain clinical conditions, especially since there are no large series in which diastolic pressure has received attention. However, we believe that these may be adduced occasionally in indirect applications to give a false sense of security. The term blood pressure, unless otherwise qualified, will be taken for systolic pressure in this paper.

It is my purpose in this article, after consideration of certain features of blood pressure observed in pregnancy, labor, and the puerperium, to emphasize the following points: That comparatively low blood pressure may be found in obstetric patients with no other evidence of abnormality in health; that eclampsia may occur with low blood pressure, notwithstanding the generally accepted belief to the contrary; and that extremely low blood pressure may be a forewarning of shock following labor, and not due to hæmorrhage.

NORMAL PREGNANCY AND LABOR

Attempts were early made to ascertain the influence of pregnancy, labor, and the puerperium upon blood pressure, yet the inaccuracies of the earlier instruments available did not permit of trustworthy data. Yet in 1884 Lebedoff and Porochjakow published their observations with a Basch sphygmomanometer. Two years later Louge, who had made some 4000 records during labor and the puerperium, concluded with Marey that the instrument of the latter did not permit of the study of the arterial tension under such conditions. The first well-known paper on kindred conditions records observations of the blood pressure in eclampsia, and considers the phenomena under normal conditions in less detail. This article was published in 1897 from the Paris Maternité by Vaquez, and his interne Nobecourt; and was followed in the next year by the paper of Vaquez and Millet, studying the normal phenomena. The Basch-Potain instrument was used in these series. Immediately following this the subject attracted fairly wide attention.

As a result of these investigations we are in the possession of many confirmed observations of the systolic blood pressure during normal pregnancy, labor and the puerperium. It appears that the blood pressure is lower during the earlier months of pregnancy than at term, as the averages of many readings during the last two months show a gradual rise. (Wiessner.) This rise is not marked, being 10 or 15 mm. Hg. on the average, and is attained about one week before labor. There is some recession from this level during the last week of pregnancy. We should expect such phenomena when we consider the alterations in intra-abdominal pressure which are caused by the presence of the growing fœtus. These may be influenced by other factors as suggested by Gnecchi, to-wit: Cardiac hypertrophy, or the effects of a very mild autointoxication. The observations of Vogeler do not support the view that there are constant differences in pressure with varying fœtal positions, nor yet from the patient's age *per se*. Differences between the pressures of multipara and nullipara also appeared as coincidental.

With the advent of labor there is an elevation of pressure not only during uterine contractions, but also in the intervals, unless the patient is in a state of exhaustion. Cook has shown that the rise during uterine contrac-

tions is not due in large part to the effect of voluntary abdominal contractions, since it appears but slightly diminished during anæsthesia. As would be expected, the painless contractions during pregnancy, termed the Braxton-Hicks contractions, cause a rise similar to that of labor pains, and differ from them only in degree. As labor progresses these features become more pronounced when the child is forced through the pelvis. The greatest rise which Vaquez and Millet recorded (between 18 and 25 cm.) occurs during the expression of the head. Extreme hypertension is sure to result from manipulations during artificial delivery through the natural passages. This is most marked during traction upon the child, either with instruments or in extraction following version. After birth, and usually immediately, the pressure falls at least to the level of that of pregnancy. Vogeler found the lowest readings by the eighth hour after delivery. There are usually no wide variations from the normal during the puerperium under normal conditions.

Quite naturally the averages of pressure readings vary considerably. Not only were the older instruments inaccurate, but some series of recent date are recorded with the narrow cuff, shown to be inaccurate by the work of Recklinghausen, Janeway, and Erlanger. All authors have not considered the modifying effect of posture, relaxation, and freedom from excitement, which may readily cause errors of more than 10 mm. Nearly all readings, moreover, have been taken by the palpatory method. The following figures, therefore, may not admit of direct comparison, yet are of relative interest:

The normal blood pressure of young adults has been variously estimated. Cook, in 1903, with the Riva Rocci narrow arm piece instrument, found it to average 115 for young non-pregnant women. Thayer, with the same instrument, attained an average of 140 mm. for 126 men between 20 and 40 who had not had typhoid. Erlanger in the same year found the average with his instrument as 110 for non-pregnant adults between 20 and 25 years. Sakurai, with the Haak instrument, a modified Riva Rocci, obtained ranges of 90 to 122 mm. Hg. for the non-pregnant women. Judging from the readings of Erlanger made with cuffs of differing sizes, there is less variation between these figures than is at first apparent. Vaquez, in 1906, announced that in his observations of ten years the systolic blood pressure of normal pregnancies ran between 130 and 150 with the Basch-Potain instrument. Fellner, 1901, with the Gärtner tonometer on the finger obtained ranges from 108 to 136. Schroeder the following year obtained higher readings with the same instrument. Goldwater, with the tonometer, obtained variations of 105 to 132 for eleven normal cases under 30 years. Vogeler, with a wide cuff Riva Rocci, writes (1907) that by carefully observing the necessary rules of technique, making the patient rest before taking pressure, eliminating the nervous causes, and ruling out cases with thickened blood vessels, nephritis, etc., he never obtained readings higher than 150 in normal cases (dorsal position). Hirst obtained an average of 118. Bailey, with the wide cuff Stanton instrument, obtained an average pressure of 118 mm. Hg. By recasting his cases we find that about 83 per cent of his 1,136 readings are under 130. There are 6.2 per cent below 100, and ½ of 1 per cent between 170 and 180. Slemons and Goldsborough, 1908, with the Erlanger instrument found average systolic pressure of 127 mm., with 74 mm. Hg. as the average diastolic pressure. Sakurai, 1909, obtained readings between 97 and 143, with an average of 118.6 mm. with the Haak instrument (palpation).

It has been my habit during the last ten years to take blood pressure readings on all private obstetric cases. During this period I have been constantly impressed with the fact that these readings were frequently lower than those generally given in the literature. The material studied comprised many women of neurasthenic disposition and of the poorly muscled physique, which are so frequently associated with generally contracted pelves. In these the pressure was invariably low, and the pulse commonly quite fast. Yet the pressure in the elderly primiparæ, of which there were ten more than 38 years of age in my

series here reported, did no more than bring up the average to normal.

Tabulating the systolic readings of 100 private cases, which have had at least 4 blood pressure determinations during their pregnancy, we find average systolic reading of 104.6 mm. Hg. with variations between 78 and 145 mm. The case giving 78 mm. will be considered later. There was one other reading below 90; viz., at 87. Grouping the pressures we find:

35.2 per cent of the entire series below 100.
43.5 per cent between 101 and 110.
13.1 per cent between 111 and 120.
8.2 per cent between 121 and 145.

I had hoped to compare with these figures the averages from a similar series taken in the hospital wards, yet this has proven impossible, as the majority of the cases enter the Presbyterian Hospital in labor. Dr. Carl Davis has kindly obtained for me the figures for 60 obstetric cases during the puerperium. The instrument was the same (Stanton), although in this series the pressure was determined by the auscultatory method, which Davis believes here gives an average of 6 mm. higher than that he would obtain by palpation. The average systolic reading was 124.6, with variations from 105 to 146 mm. It is of interest that the lowest pressure in his series was taken from a patient of mine on whom I obtained readings during pregnancy of 100, 105, 108, and 105 by palpatory method. Grouped according to percentages, we find the averages of these cases far higher than mine, even allowing 10 mm. as the difference in methods. The table is as follows:

Below 110 mm	12 per cent of 60 cases
111 to 120 mm	32 per cent of 60 cases
121 to 130 mm	28 per cent of 60 cases
131 to 140 mm	25 per cent of 60 cases
Above 140 mm	4 per cent of 60 cases

I am unable to explain the differences between these series save on the ground of Brunton's teaching. I conclude, therefore, that the ward cases were more vigorous and physically stronger individuals.

ECLAMPSIA

Following the report of Vaquez and Nobecourt in 1897, many others have called attention to the presence of high blood pressure in this disease. It is natural, therefore, that this phenomena should be emphasized as a premonitory sign of value. Vaquez, writing in 1907, after ten years' experience with blood pressure in obstetrics, voices the extreme view. He states that if blood pressure is very high, convulsions will follow, claiming that eclampsia seizures will never occur in cases of normal tension. Vogeler states that it is impossible to find in the literature a case of moderately high tension, which was carefully watched, and in whom the blood pressure was carefully taken which went on to eclampsia.

The majority of reviews, in this country at least, have accepted these teachings, especially in the light of the multitude of cases of eclampsia in the literature with extremely high blood pressure. While there is no doubt that eclampsia is usually signaled by a rising blood pressure, eclampsia may occur with normal pressure. The statements quoted above may be misleading, and the means of producing false security. The interpretation of blood pressure readings is not always simple. Much depends on the time and conditions when taken. Readings during labor are most apt to be unreliable. The unusual emphasis placed upon hypertension as a certain sign of toxæmia is largely due to the fact that arteriosclerosis is most uncommon during the child-bearing age. Yet Russell's work on arterial hypertonus, sclerosis and blood pressure in 1908 should make us cautious in ascribing all hypertensions to toxæmia. The contentions of Vaquez, Beau, Vogeler, and others, that blood pressure of 180 or more during pregnancy means toxæmia is not supported by the observations of Slemons and Goldsborough. The latter authors report the case of a IX-para who gave no evidence of toxæmia yet who several times had systolic pressure of 180 mm. Hg. with the Erlanger instrument, and an average pressure of 169 mm. during the last days of pregnancy. Green, in 1912, from a study of his cases, came to the conclusion that the height of the blood pressure is proportional to the severity of the case with toxæmias. On recasting his toxæmias with chief symptoms of albumin we obtain the following:

One case with blood pressure 130, albumin 1. %
One case with blood pressure 140, albumin 0.5 %
One case with blood pressure 140, albumin 0.7 %
One case with blood pressure 150, albumin 0.25%
One case with blood pressure 160, albumin trace
One case with blood pressure 175, albumin 0.25%
One case with blood pressure 180, albumin 0.3 %
One case with blood pressure 190, albumin 0.25%
One case with blood pressure 190, albumin 0.3 %
One case with blood pressure 210, albumin 1. %

From which we see it is not necessarily proportional to the amount of albumin.

I have in my own experience twice met with eclampsia developing in women on whom blood pressure reading during pregnancy had been found normal.

The first case was reported to the Chicago Gynecological Society February 19, 1909, as an example of eclampsia, which developed in a woman under careful observation, who had given no premonitory symptoms.

A multipara of 32 years. Nearly continuous nausea for three months without much vomiting, followed by good health, save for continuous indigestion during the last three months. Soda gave relief and was freely taken. One time I was quite startled by the patient's statement that she had just finished the second pound of soda taken for nausea during the last few months. Four blood pressure readings scattered through pregnancy ranged from 90 to 105 mm. Hg. Stanton wide band instrument; palpatory method. At the onset of labor, pressure was 113. The urine had been followed for albumin and casts daily for the last month, with negative findings. The twenty-four hour weekly specimens ranged in amount from 2,000 cc. to 3,800 cc., with 3,750 ccs. two days before labor. Labor began eighteen days before calculated term. After ten hours of very easy first-stage pains there was a single convulsion. There was no evidence of œdema. The pulse had felt soft and normal throughout the labor. Immediate delivery with forceps, easy extraction, chloroform anæsthesia. Four hours later the blood pressure was 93, and on the following morning 120, and 125 on discharge fifteen days later. Three nitrogen determinations made during the last weeks in pregnancy gave normal variations of total nitrogen and ammonia nitrogen.

The second case was a nullipara, thirty-seven years of age. The blood pressure during pregnancy was 110 on two determinations. During the first stage of labor between pains it was 125. Labor was normal, the first stage of sixteen hours, second stage of two hours. Child was spontaneously expelled in L. O. A. Chloroform was given as the head came over the perineum. A convulsion came on without warning two hours after birth, and one hour later there was another. The blood pressure two hours after the second convulsion was 135, and on the following day 125. The urine had been examined daily for casts and albumin during the last month of pregnancy, always with negative findings.

In addition to these, we may add a third case reported to the Chicago Gynecological Society March 24, 1911, as eclampsia without convulsions.

A III-para, 35 years of age, with no history of toxæmia conditions in any of her three pregnancies. During the last six weeks of her fourth pregnancy there were some signs of toxæmia. Dr. Loeb found no casts, nor œdema, nor cardiac complication, but enjoined rest in bed because of $\frac{3}{4}$% per cent albumin and diminished urine. It appears that the patient may have denied subjective symptoms, wishing to avoid the induction of labor. Nor did she keep to her bed. Six weeks after the first symptoms she fell into labor during which she complained of intense epigastric pain. Shortly after, she became comatose without convulsions. The child was delivered immediately without anæsthesia. Three hours later we found a rather soft pulse, about 120, blood pressure of 100 and temperature of 100½° F. A similar reading was taken an hour later. Death occurred 13 hours after the onset, with epigastric pain, preceded by anuria and a gradual rise in the pulse rate. The examination at the time the blood pressure was taken did not indicate shock.

We have also seen one case with a blood pressure reading of 85 mm. taken 22 hours after the onset of eclampsia, with immediate delivery, followed by shock and hemiplegia.

Similar instances are not unknown in literature. Little, in 1908, reports a series of 40 eclampsias. The blood pressure was elevated between 160 and 200 in 37 cases, yet in three cases was as low as 125, 133, and 150 mm. Hg. respectively. We believe we may also adduce one case of Vogeler's as an example of eclampsia occurring during medium blood pressure during labor, notwithstanding the fact that Vogeler classifies it under hypertension. The case was one of four which Vogeler states presented extreme hypertension, and is of interest in that he thus comments upon it. Of these cases, "one only developed convulsions, and was the case of lowest blood pressure in the group." The history shows that the patient was completely examined 3 days before convulsions, and the urine was examined and found to be negative. Two days later she complained of a gastro-

intestinal upset and was given a cathartic. The night following that day she awoke from sleep at 2 A. M. with a violent headache and epigastric pain. At 7 A. M. she became blind, etc. Blood pressure at 9:45, 10:15, and 10:45 A. M. recorded 155. At 10:55 there was a convulsion, and the blood pressure attained 182 mm. Hg. The cervix was dilated to two finger-breadths. Immediately after accouchement forcé, under chloroform, blood pressure was 80, rising shortly to 100, and ranging between 130 and 150 for some four or five days. We submit that this case, presenting a systolic pressure of 155 while in labor, cannot be adduced as an example of extreme hypertension by one who classifies normal pressures 100 to 150, moderate hypertension under 180, and extreme hypertension above 180. The case is merely one other instance of the type of true eclampsias so difficult to recognize until the presence of subjective symptoms.

Bailey, in 1911, reported two cases of profound fulminating toxæmias with low blood pressures. Both cases were admitted through police headquarters by whom they had been regarded as alcoholism. The first case had blood pressure of 70 mm. just before delivery. Eighteen ounces of urine were obtained in 14 hours. Death followed 19 hours after delivery.

The second case also had repression of urine, 13 ounces being the greatest amount in any day under admission. Blood pressure was 75 before delivery, and 80 mm. two days later. Death took place five and one half days after admission.

Bailey concluded that convulsions usually occur when the blood pressure is in the neighborhood of 200, but may occur as low as 155; also that convulsions do not occur when the blood pressure is lowered by poor resistance or by drugs producing collapse.

We believe, as a result of our investigation, that while eclampsia generally gives warning of its approach by a rise in the blood pressure of the individual, the disease may occur without warning; that we are forced to admit the possibility of eclampsia outbreaks, without warning of urinary or blood pressure phenomena.

LOW BLOOD PRESSURE AND SHOCK FOLLOWING LABOR

In the 1897 edition of Lusk's splendid treatise on Midwifery, there is an article of nearly two pages on a subject which frequently receives but as many lines in works of more recent date. Indeed, one may often find scant mention of shock following normal spontaneous labor not associated with hæmorrhage. Lusk in his chapter on "Nerve Exhaustion" and shock thus comments: "Twenty years ago these pathological states played a conspicuous part in the etiology of sudden death during childbirth. Now the fashion has changed. . . . None the less, the need remains to account for a class of cases in which death takes place without recognizable organic lesions. . . . In the absence of visible lesions or the characteristic symptoms of the conditions to which death is usually referred, we have the right to attribute the melancholy issue to the same causes which, outside of childbed, produce identical phenomena." With this preamble there follows a vivid description of the shock which has been met with by nearly all men who have had large series of cases in this country. As a hospital assistant, I well remember my chief's description of two cases of shock from causes that remained unexplained unless attributable to the labors. There was no hæmorrhage in either case. A few years later I saw a similar case in consultation. Quite recently there came under my care a patient who presents the following most remarkable history:

A young woman of 22 years was seen by me in the spring of 1910 when five months pregnant. The family history was negative. Measles and chickenpox were the only diseases in the patient's life. The examination disclosed no abnormalities. The patient's color appeared so good that the blood examination which I make as a routine was omitted. Blood pressure was 95. The patient was living in a neighboring city and was not seen again until the week before labor. The urine had been examined daily during the last month with negative results for albumin, casts, normal amount, and specific gravity. There was no constipation, nor yet looseness of the bowels. Labor began June 19, 1910, at 3 A. M., but did not settle down until morning, so that she did not enter the hospital until 7:30 A. M. The first stage was in no way unusual. The membranes were

ruptured artificially at 5 P. M. Second stage pains were at three-minute intervals. Birth of a 3,600-gram child in R. O. A. was without further incident. No anæsthesia was given save a few whiffs of chloroform at the moment of making a small episiotomy. Two catgut sutures and two silkworm-gut sutures were used for closure, without anæsthesia. The various stages of labor were as follows: First stage, 14 hours; second stage, 1 hour 7 minutes; third stage, 30 minutes; total, 15 hours and 37 minutes. The labor must be classed as easy. There was scant blood lost following the Schultze separation of the placenta. One dram of ergot was given by mouth. During labor the temperature at 7:30 A. M., 10 A. M., and 12 P. M. was 98.8, 98.6 and 99.2 respectively, with pulse of 92, 120, and 88 at the same periods. The fœtal heart rose from 35 to the quarter minute to 39 or 40 shortly before the onset of the second stage. One hour after the birth of the child the patient became shocked, the pulse began to be irregular, rose to 120, then shortly ascended to 150 and 160, and remained elevated at 150 during the night. There was no external hæmorrhage, and the uterus remained firmly contracted. Water and stimulants had been given, and the patient voided eight ounces twelve hours after delivery. The recovery from the shock consumed several days, as the pulse was 132 nearly twenty-four hours after labor. The respiration ranged from 18 to 24. About the eighth day the temperature rose between 100.5° and 101°, while the pulse, which had remained above 100, again ascended. The blood pressure was 88 as taken by a hospital interne. The hæmoglobin was 30, leucocytes 20,000 and red blood cells 2,080,000. The lochia now became more red, and it appeared as if something had been retained in the uterus after labor, although the placenta had appeared intact. The uterus was explored on July 5th without finding cause for the discharge. One week later the curette was used again and several pieces of placental tissue the size of rice bodies were removed from the left uterine cornua, together with a thickened and œdematous endometrium, with marked proliferation of the glands. Following this, recovery was gradual, although the hæmoglobin did not reach 70 until late fall. During the winter she attained her usual health and the hæmoglobin ranged about 85.

One year later the patient returned to me when four months pregnant. The blood pressure was 78 and Hb. 85. There had been comparitively little nausea. I sent her to an internist stating that I wished an opinion as to the condition of the heart and blood vessels, which appeared to me unlikely to stand the strain of labor, and that I was willing to abort her for that reason. The blood pressure reading was confirmed, but the internist thought that with care the patient could be put in shape for labor; that from the reasons given he could not agree to abortion. Tonic treatment was given. Three months later her blood pressure was found to be 75, pulse 96, and Hb. 80. Varicose veins on her legs now appeared, also dilated capillaries on the face and hands, for which she was sent to Dr. Ormsby for consultation. Three weeks later the blood pressure was 82, pulse 96, Hb. 85. A few days before labor the systolic blood pressure ranged between 85 and 90 on several examinations. The membranes ruptured on February 18, 1912, at 2 A. M., following which there was no pain for two days with the exception of a few feeble contractions. The patient was able to sleep. The following night pains reappeared, disappearing after a few hours. The temperature had now risen to 101.2°, and pulse frequently reached 120. A small sized Voorhees bag was inserted without anæsthetic at 4 A. M., February 20th. Pains shortly followed. At 8 o'clock the child was born after an easy second stage of less than one hour's duration.

Anticipating shock, the arms and legs were bandaged before the time of delivery; coffee and digitalis were given immediately after delivery. At that time the pulse was 108, temperature 100, respirations 20. Thirty minutes after labor, before the placenta had been separated and before an effort had been made to express it, she fell into shock and the pulse ascended to 160, immediately following which it could not be felt at the wrist for four hours. During this time she seemed barely alive, making feeble response to most vigorous stimulation. At 3 o'clock in the afternoon the pulse remained at 160. During the night it ranged about 140, with some vomiting. Respirations were about 32. The following day the pulse did not go below the level of 120, the respirations from 28 to 34. We had thus far made no attempt to remove the placenta, feeling certain that such effort would result only in immediate death. Forty-eight hours after delivery the placenta was removed without anæsthesia. This operation was followed by a chill and a pulse which again disappeared at the wrist and ranged, six hours after the removal of the placenta, at about 150. In spite of the fact that there had been extremely little loss of blood at the time of the labor, and very little oozing during the time the placenta remained in the uterus, hæmoglobin on the fourth day was read at 40 per cent, and on the eighth day 30 per cent. The puerperium was now complicated by an infection and thrombosis of the external iliac vessels.

After a long, stormy convalescence, interrupted at the very end by acute bronchitis, she was discharged on the 74th day following labor. The hæmoglobin was 60 per cent and R. B. C. 3,750,000.

Careful study of this case, we believe, impresses the importance of the diagnostic value of a low blood pressure as a sign of the patient's inability to stand the strain of labor. It appears from study of the literature that there are two types of low blood pressures, one of which appears to be compatible with good health; while the other generally follows

some acute infection or develops during early phthisis. Lauder Brunton has recently emphasized that the feelings of the patient with low blood pressure varies. While some work well and easily with blood pressure between 90 and 100, others tire and are incapable of prolonged manual work. He believes that rest in bed with diet and cardiac tonics is indicated when the pulse falls below 80 or 90. While many of my patients with blood pressures of 90 have passed through normal labor, I believe that all cases with pressure below this limit should be subjected to the most careful scrutiny. If this condition is associated with low hæmoglobin, there is no doubt in my mind that the prognosis is serious as a rule, as experience has convinced me that it is a difficult matter to raise the hæmoglobin or blood pressure during pregnancy. It seems highly probable, therefore, that through the study of blood pressure we may have a forewarning as to the possibility of a complication fortunately rare at the present time; i.e., shock following labor not associated with or resulting from hæmorrhage. It is extremely regrettable that my case was not studied as to pulse pressure. Only systolic pressures had been taken by those who studied the case.

BIBLIOGRAPHY

BADGER. Some Blood Pressure Observations in Eclampsia. Boston M. & S. J., xcviii, 607.

BAILEY. The Blood Pressure Index of Eclampsia. Surg., Gynec. & Obst., 1911, xiii, 505.

VON BASCH. Ueber die Messung des Blutdrucks am Menschen. Ztschr. f. klin. Med., 1981, ii, 79.

COOK AND BRIGGS. Clinical Observations on Blood Pressure. Johns Hopkins Hosp. Rep., 1903, xi, 451.

DAWSON. Systolic Output and Work of the Heart, and their Relation to the Blood-Pressures. Brit. M. J., 1906, ii, 996.

EDGECOMBE. Low Blood Pressure. Practitioner, Lond., 1911, April.

ERLANGER. A New Instrument for Determining the Minimum and Maximum Blood-Pressures in Man. Johns Hopkins Hosp. Rep., 1904, xii, 53.

ETTINGER. Die Auskulatorische Blutdruckmessung nach Korotkow. Wien. klin. Wchnschr., 1907, xx, 992.

FELLNER. Herz und Schwangerschaft. Monatschr. f. Gynäk., 1901, xiv.

FISCHER. Die Auskulatorische Blutdruckmessung im Vergleich mit der Oszillatorischen von Heinrich von Recklinghausen, etc. Ztschr. f. diätet. u. physik. Therap., 1909, xii, 389.

GITTINGS. The Auscultatory Blood Pressure Phenomenon. Arch. Internal. Med., 1910, vi, 196.

GOLDWATER. Notes on Blood Pressure in Man. Med. News, 1903, lxxxii, 926.

GOODMAN and HOWELL. Auscultatory Method of Determining Blood Pressure. Am. J. M. Sc., 1912, Sept.

GREEN. Blood Pressure in Toxæmias of Pregnancy. Boston M. & S. J., 1910, Apr. 28; 1912, Feb. 1.

HIRST. N. Y. M. J., 1910, June 11.

JANEWAY. The Clinical Study of the Blood Pressure. N. Y., 1904.

KOROTKOW. Zur Methodik der Blutdruckmessung. Mitt. d. k. mil.-med. Akad. zu St. Petersb., 1905, xi, 365.

LANG and MANSWETONA. Zur Methodik der Blutdruckmessung, usw. Deutsche Arch. f. klin. Med., 1908, xciv, 441.

LEBEDOFF UND PORACHJAKOW. Basch's Sphygmomanometer und der Blutdruck während der Geburt und des Wochenbettes, usw. Zentralbl. f. Gynäk., 1884, viii, 1.

LITTLE. Treatment of Puerperal Convulsions. J. Obst. & Gynec. Brit. Emp., 1908.

MAREY. Trav. du Lab., 1876, ii, 1309.

POTAIN. La pression artérielle de l'homme. Par., 1902.

QUEIREL. De la valeur prognostique de la tension arterielle chez la femme enceinte. Ann. de gyn. et d'obst., 1908, Dec., 727.

QUEIREL ET REYNAUD. Tension artérielle et puerpéralité, grossesse, accouchement, suites de couche, physiologiques et pathologiques. xiii Cong. Internat. de med., Par., 1900, xv, Sect. d'obst., 170.

RECKLINGHAUSEN. Ueber Blutdruckmessung beim Menschen. Arch. f. exper. Path. u. Pharmakol., Leipz., 1901, xlvi, 78.

RECKLINGHAUSEN. Unblutige Blutdruckmessung. Ibid., 1906, 55, 375.

RUSSELL. Arterial Hypertonus, Sclerosis and Blood Pressure. Phila., 1908.

SAKURAI. Blutdruckmessungen bei Schwangeren, Gebärenden, und Wöchnerinnen. Beitr. z. Geburtsh. u. Gynäk., 1909, xix, 498.

SCHRÖDER. Weitere Untersuchungen über das Verhalten des Blutdrucks in der Schwangerschaft, usw. Beitr. z. Geburtsh. u. Gynäk., Festschr. f. Fritsch, 1902. Quoted from Jaschke.

SLEMONS AND GOLDSBOROUGH. Die Geburtsh. Bedeutung des Blutdrucks, usw. Zentralbl. f. Gynäk., 1908, 21.

VAQUEZ ET NOBECOURT. De la pression artérielle dans l'éclampsie puerpérale. Bull. et mém. Soc. med. hôp. de Par., 1897, xv, 117.

VAQUEZ ET MILLET. Du cour dans la grossesse normale. Presse méd., 1898, vi, 61.

VAQUEZ. De la tension artérielle pendant la grossesse et les suites de couches. Bull. Soc. d'obst. de Par., 1906, séance Feb. 15th.

VAQUEZ. Valeur diagnostique et prognostique de l'élévation de la pressure artérielle au cours de l'éclampsie puerpérale. Bull. Soc. d'obst. de Par., 1906, ix, séance Feb. 15.

VIERORDT. Die Lehre vom Arterienpuls in Gesunden und Kranken Zuständen. Braunschweig, 1855.

VOGELER. Blood Pressure During Pregnancy. Am. J. Obst., N. Y., 1907, lv, 490.

WARFIELD. Studies in Auscultatory Blood Pressure Phenomena. Arch. Internal Med., 1912, x, 3, 258.

WIESSNER. Ueber Blutdruckmessungen während der Menstruation und Schwangerschaft. Gesellsch. f. Geburtsh. u. Gynäk. zu Leipz., 1899, June 19th; Zentralbl. f. Gynäk., 1899, 1335.

PATHOGENESIS OF CONGENITAL CYSTIC DISEASE OF THE PARENCHYMATOUS ORGANS

By ARTHUR E. HERTZLER, M. D., Kansas City, Missouri

UNDER the name of congenital cystic disease has been described a condition characterized by a collection of cysts varying in number, size and contents, found in several of the parenchymatous organs and occurring both in the earliest periods of life and in later ages. These cysts have been noted most frequently[1] in the kidneys. They also occur in the liver, pancreas, spleen, testicles, ovaries, cerebral appendages and broad ligaments. Although they have much in common, irrespective of the organ or tissue in which they occur, a different genesis has been ascribed to them in each situation.

I have reviewed the existing views as to their nature and origin and have examined a number of specimens of these and allied conditions. In order to determine the properties which they have in common, I shall consider their structure and development in each organ separately, and then compare the data thus gained with a view to suggesting the possibility of a common origin.

Kidney. Because of the vastly greater number of cases reported in the kidney and because nearly all the literature has to do with the disease as affecting this organ, it is possible to discuss it with some completeness.

Age. Like the solid tumors of the kidney, cystic tumors occur at two distinct periods, early life and middle age. Sieber divides the groups at the 20th year. Below this age he reports 32 and after this age 212. Of the 32 occurring before the 20th year, 22 occurred before the 6th year. This corresponds very closely with the age incidence of solid tumors as presented by von Bergmann. The first period corresponds to the embryonal group of tumors. In the second period the disease is found frequently between the 40th and 50th years and half of all cases occur between the 40th and 60th years. This corresponds very closely to the occurrence of the so-called hypernephromata in middle life.

Causation. With reference to causation the age incidence is suggestive. The youngest living case reported is 8 weeks. A case of still-born was reported by Theilhaber.[2] Then 3½ and 6½ months (Savory) and 7 months[3] are the youngest. The occurrence of such gross pathological changes at such an early age naturally has given rise to the supposition that the disease in all instances is congenital or is an anomaly of the developmental period. Why a long period intervenes before the second age of frequency has been explained neither for the solid nor for the cystic tumors.

Explanations of cystic disease have been for the most part based upon the assumption of retention cysts, or retention cysts with neoplastic development. The manner of the occlusion of the outlet has been the chief subject of controversy. In the discussion of this problem the uriniferous tubules alone have received attention. Being retention cysts the contents was assumed of course to be urine. It may be stated in passing that the mere fact that cystic contents in other organs is like that of the kidney has not up to date caused any doubt as to the urinous nature of the contents in the kidney cyst. The problem being greatly simplified by this assumption, it remained only to arrive at some acceptable explanation for the occlusion. Unfortunately for harmony, investigators have been more fastidious in this than in the other phases in the problem and a vast literature has arisen concerning it. The earliest explanation was a stricture of the urethra.[4] After this had been disproven[5] because it was pointed out that while hydronephrosis might be so produced a dilatation of the tubules was out of the question, the uriniferous

[1] Sieber: reports 212. Deutsche Ztschr. f. Chir., 1905, lxxix, 407.

[2] Monatschr. f. Geburtsh. u. Gynäk., 1899, ix, 496.

[3] Borst: Festschr. zur Feier des 50 jährigen Bestehungs der physik. med. Gesellsch. zu Würzburg, 1899, 7.

[4] Herz: Virchow's Arch. f. path. Anat., 1865, xxxiii, 232.

[5] Orth. v. Mutach: Virchow's Arch. f. path. Anat., 1895, cxlii; Senator, in Nothnagel, Spez. Path. u. Therap., bd. xix, 1899.

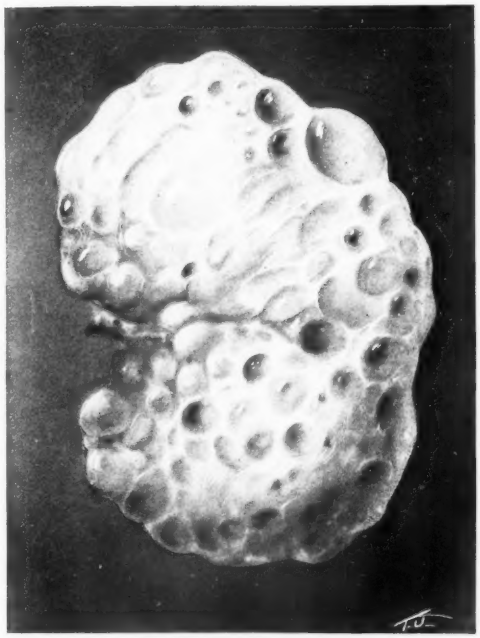

Fig. 1. Polycystic kidney.

tubules themselves were supposed to have been occluded. Frerichs, Albers and Ruyssenaers assumed a fibrinous exudate. Johnson and Rokitansky assumed a fibrinous débris the result of desquamative nephritis. Virchow[1] assumed an occlusion by concretions of uric acid. He later ascribed the occlusion to the contraction of interstitial connective tissue. This theory was formulated to apply to cystic kidneys of the new-born, but was later extended to include those occurring in the adult as well.[2]

A more recent retention theory is based on the assumption of error in development. A portion of the tubule develops from the kidney pelvis and the other portion from the glomerulus. The failure of these two portions to unite leaves a blind sac in the distal portion. Based on these embryological considerations Koster[3] supposed that the cysts were collections of fluid in the blind portion.

[1] Berl. klin. Wchnschr., 1892, xxix, 105.

[2] Thorn: Beitr. zur Genese der Cystenniere. Inaug. Diss. Bonn, 1882.

[3] Niederlandsch Arch.voor Geneesk. en Natuurkunde,ii, p. 774, quoted by Theilhaber.

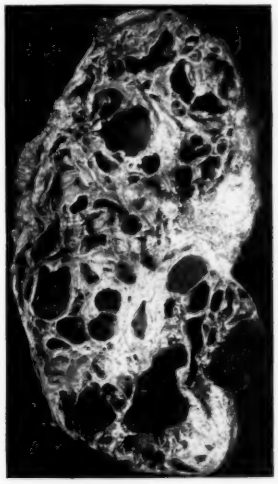

Fig. 2. Cross section of polycystic kidney.

When we consider the validity of the retention theory the absolute lack of evidence is at once apparent. The hypotheses of occlusion by albuminous material, concretions and interstitial inflammation become at once untenable when it is noted that in many of these cases the kidney substance between the cysts is quite normal. The developmental theory is dubious inasmuch as the develop-

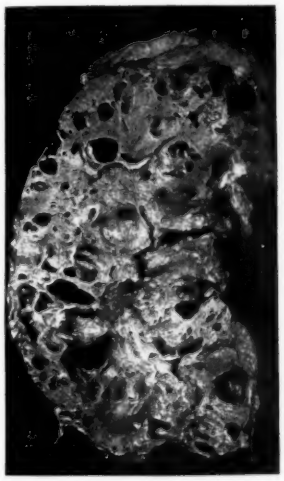

Fig. 3. Cross section of polycystic kidney.

Fig. 4. Polycysts of the kidney. (*a*) Cuboidal cells. (*b*) Irregular cells. (*c*) Flat cells.

Fig. 5. Polycysts of the kidney. (*a*) Cuboidal cells. (*b*) Flat cells. (*c*) Renal tubule.

Fig. 6. Endothelium of polycysts of the kidney and adjacent kidney tubules.

Fig. 7. Walls of a polycyst of the kidney with thick flat cells.

ment of the tubules in two sections is disputed[1] and if this were the method of development there is no evidence whatever pointing to a failure of union between the two segments. Against the theory of occlusion of a tubule stands the fact that the cysts are spherical. If the occlusion of a tubule were the cause, the cavities should be tubular in the early stage. If they were developed from the occlusion of the cortical segment the cysts should be confined to the cortical portion of the kidney. On the contrary the cystic disease of the kidney, even in the new-born, shows all portions of the kidney equally affected. If there were such failure to unite there is no evidence that the cystic contents is the result of secretion of the renal epithelium.

The second theory is that the cysts represent neoplastic formation. This view is accepted by a representative number of recent observers, notably Albarran and Imbert. That active processes are at work is obvious from the fact that papillary formations occur within the cysts, which is evidence that the epithelium possesses a proliferative as well as a secretory activity.

[1] Gerhardt: Arch. f. mikr. Anat. u. Entwickelungsgeschichte, 1901, lvii.

Fig. 8. Polycysts of the kidney showing mixed cells at (*c*) and flat cells at (*a*) and (*b*).

Fig. 9. Endothelium of a polycyst of the kidney with adjacent fibrous tissue.

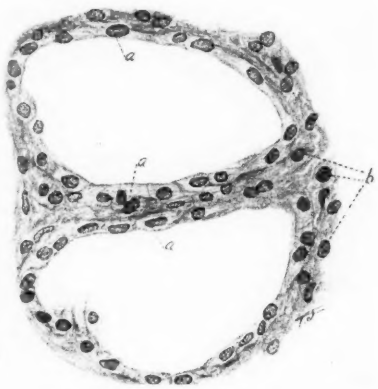

Fig. 10. Polycysts of the kidney. (*a*) Flat cells lining the cysts, and (*b*) large cells on the interstitial tissue.

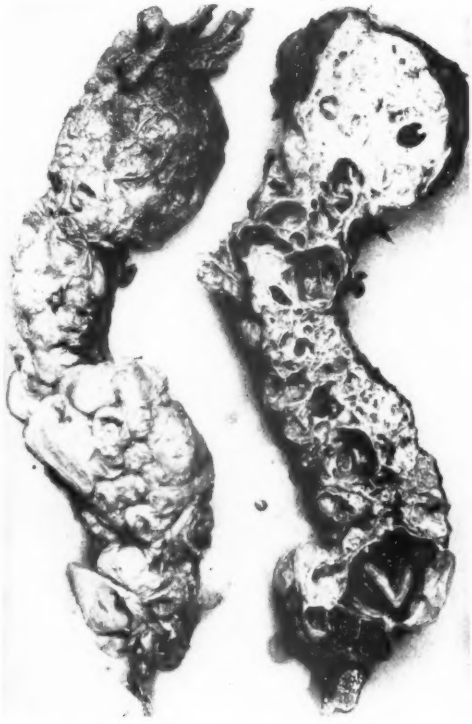

Fig. 11. Polycystic disease of the pancreas. (*a*) External surface. (*b*) Cut surface. (Photograph loaned by Dr. J. W. Perkins).

Sabourin[1] first ascribed an active rôle to the kidney epithelium, observing that it loses its character as renal epithelium and begins to proliferate. Cornil and Brault[2] assumed that the renal epithelium undergoes a change and secretes colloid material. These changes may come according to them as a result of chronic interstitial change or without it. Hommey disregards the interstitial changes entirely and accepts the cystic as primary. Lejars[3] comes to the same conclusion inasmuch as similar processes are observed in other organs. Von Kahlden[4] accepts them as neoplastic processes, having confirmed the work of Nauwerck and Hufschmidt who regard them as adenocystomata. The leucocytes according to these authors may furnish the first cystic contents together with colloid degeneration of the epithelial cells. The first filling of the small cysts may come from the blood-vessels, and in later stages, according to them, transudation from this source plays the chief rôle.

[1] Arch. de Phys., 1882.
[2] Études sur la pathol. des reins, Paris, 1884.
[3] Thèse, Paris, 1888.
[4] Beitr. z. path. Anat., 1894, xv, 626.

Fig. 12. Polycystic disease of the pancreas. Normal pancreas at the right; small polycysts at the left of the specimen.

The neoplastic theory is the only one deserving serious criticism. That the cysts are more than retention cysts is obvious from the nature of their walls and the progressive character of their development. That the

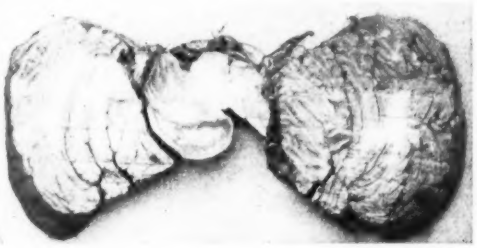

Fig. 13. Cyst in cerebello-pontine angle in polycystic disease of the pancreas (Dr. Perkins' case).

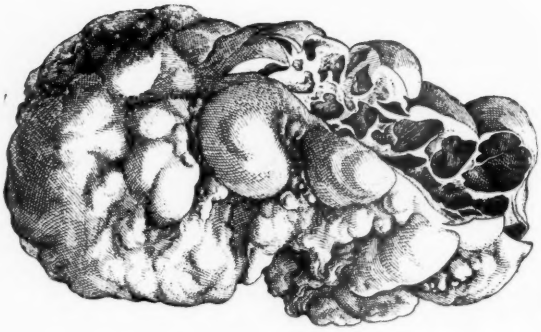

Fig. 14. Endothelium wall of lymph cyst of spleen (Schmidt).

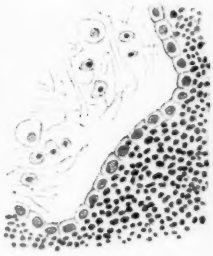

Fig. 15. Pararenal cyst (Lejars).

Fig. 16. Cystic lymphangioma of the forearm.

contents is not urine is obvious because of its composition and its similarity to the contents of cysts of the liver and pancreas. But while the neoplastic theory may be admitted, the origin of the cysts rests on an uncertain basis. Nowhere have they been proven to have a direct relationship with the uriniferous tubules. Adenomatous proliferation of uriniferous tubules bears no resemblance to polycystic disease. That renal epithelium may undergo a change whereby it is enabled to produce a colloid material likewise is unproven.

Structure. Polycystic kidneys usually present large lobulated masses many times the size of the normal kidney. Individual cysts project from the surface as hemispherical translucent vesicles (Fig. 1). They vary from those barely visible to the size of an orange. On section the kidney is filled more or less with cysts, corresponding to the size of the external protuberances (Fig. 2). In some regions normal kidney structure remains about the cysts, while at others the entire field seems to be occupied by the cysts (Fig. 3). Often surrounding the cysts is a layer of connective tissue showing narrow clefts within it. In some instances the kidney tissue shows fibrous proliferation.

The microscopic study of the cyst wall shows a layer of fibrous tissue lined by a layer of cells varying in height. The literature speaks of these as being flat, cuboidal and even columnar, though the published cuts nowhere give any evidence of the exact form of these cells. I have studied fifteen kidneys with reference to the form of the cells lining these cysts. I have confirmed in a measure the descriptions in the literature though I have not seen cells that could be described as columnar. In some areas they may be spoken of as cuboidal. At *a*, in Fig. 4 the cells are cuboidal, at least they are as high as broad and the dividing lines are fairly distinct. At *b*, in the same figure some of the cells are, like those just referred to, irregularly cuboidal but others are broader than high, and the long axes of the nuclei are directed parallel with the surface which they cover. At *a*, Fig. 5, the cells are irregularly cuboidal while at *b*, they are flat as described at *b*, Fig. 4. In Fig. 6, at *a*, the cellular layer is relatively thick and there is no dividing line between the cells but the cells are broader than high and the large oblong vesicular nuclei are parallel to the surface upon which they lie. Fig. 7 likewise shows a thick layer of cells of the same general character as those just described but the nuclei are smaller and stain more deeply. In Fig. 8, at *c*, the same general character of cells is present, but they are more irregular and in the upper side of

the cyst there is a disposition toward piling up. At *d*, Fig. 4, and *a*, Fig. 9, where the cell layer is teased loose from its base, the ovoid flat vesicular nuclei can be clearly made out. In Fig. 10 the cells are viewed obliquely. The section represents two small cysts cut near the base. The cell outlines can be made out and the nuclei are like those in the preceding section. Cells closely resembling those lining the cysts are seen in the connective tissue about their base.

The preponderating cell type in all these sections is flat, that is, the transverse diameter is greater than the perpendicular.

Surrounding the cyst walls proper, sometimes within them, are masses of lymphocytes often so abundant as to suggest miniature lymph glands. The cyst contents are colloidal with a network of fibrils containing lymphocytes in small numbers or occasionally cells desquamated from the walls. These are usually spherical with an abundance of palely staining protoplasm and are œdematous. In this situation are often found endothelial-lined clefts. The kidney tissue about the cysts may be entirely normal, particularly in infants. In adults, particularly in those dead from uræmia, the usual structure of interstitial nephritis is common.

Liver. Polycystic disease of the liver presents the same histological appearance as that of the kidney, though the cysts in the liver are usually smaller and fewer in number than in the kidney. The bile capillaries may be found unchanged near the walls of the cysts. The cysts occur in the interlobular tissue and the surrounding tissue presents a normal appearance or the usual picture of an interstitial hepatitis. The cysts of the liver have not involved a sufficient extent of the organ to give rise to any clinical symptoms.

There has been little speculation as to the origin of these cysts. By general agreement polycystic disease of the liver has been ascribed to the occlusion of the bile ducts, though no attempt has been made to adduce any evidence to support this view. In harmony with this notion the contents was assumed to be bile. Notwithstanding that the contents is identical with that of the kidney cysts, no one has suggested that liver cysts contain urine, this too despite the fact that polycystic livers are usually associated with like disease in the kidneys.

Pancreas. Pancreatic polycystic disease is so rare that an adequate account of its structure is not recorded. In the specimens I have been privileged to examine the structure is identical with the kidney cysts and the contents are the same (Fig. 11). In the recorded cases no mention is made of the state of the adjacent pancreatic tissue. In no instance has there been any evidence of disturbance of pancreatic function. Both the gross appearance and the microscopic picture are identical with polycystic kidney (Fig. 12). No relation between pancreatic tubules and cysts can be made out in any part of the specimens. The structure of the larger cysts likewise resembles that of the kidney. There is no evidence that they contain any of the specific ferments of the pancreas. Connolly[1] reports a case in a child aged 14 months. Thiroloix and Pasquier[2] report a case in a woman aged 93. They describe the cyst as being lined with embryonal cells.

Single cysts and pseudo-cysts of the pancreas are quite common and there is a large literature concerning them. What their relation may be to the polycystic disease has not been discussed. It is interesting to note in this connection that in many of the larger cysts reported it was mentioned incidentally that there were other smaller cysts present.[3] It is of interest to note that pancreatic cysts, even the so-called true cysts, are often preceded by a violent traumatism to the upper abdomen. Lymphangiomata often have a similar etiology. To discuss this question adequately would lead me too far afield and I can only say in passing that morphologically the large cysts have much in common with the polycystic disease, but the chemistry presents conflicting evidence.[4]

[1] Lancet, Lond., March, 1911.

[2] Bull. de la soc. anat., 1892, 311; quoted by Nimier, Rev. de Chir., 1893, 764.

[3] Fitz: Am. J. M. Sc., 1900, cxx, 184; Wolff, Beitr. z. klin. Chir., 1911, lxxiv, 487; Hartmann, Cong. de Chir., 1891; Thiroloix and Pasquier, Bull. de la soc. anat., 1892, p. 312; Nimier, Rev. de Chir., 1893, 764.

[4] Körte: Die chirurgischen Krankheiten des Pancreas. Enke, Stuttgart 1898 p. 61.

Rare cysts. Cysts occurring in other regions, as in the testicles, the broad ligament and along the ureters, associated with the renal lesion are likewise similar in structure and cystic contents so far as the recorded cases present any evidence. Those of the testicle have not been separated from definite mixed tumors. Those occurring in the ureter and in the broad ligament have not been subject to histological study. Those occurring in the brain have been referred to only incidentally.[1] Owing to the infrequency with which cysts in the choroid plexus occur in association with polycystic disease and their frequency independent of any disease a degree of caution must be observed in their interpretation.[2]

That the polycystic disease is related to neoplastic processes is demonstrated by the proliferation of the cyst walls to form projections within the cavity, both epithelium and fibrous walls taking an active part in the process. That the disease is based on congenital disturbance is supported by its early and bilateral occurrence and by the frequent association of the kidney lesion with like lesions in other organs, and its disposition to hereditary association. Often other lesions of congenital origin, such as harelip and dermoids, are present in the same individual or in other members of the same family. This disposition to hereditary influences is even more strongly emphasized by the occurrence of polycystic renal disease in several members of the same or successive families, as in the cases reported by Steiner.[3]

From the foregoing we may deduce the fact that the disease is due to some congenital defect and is neoplastic in character. From the fact that the disease is the same wherever it occurs, we may assume that the cysts are derived from some tissue common to all these situations.

COMPARISON WITH CYSTS OF KNOWN ORIGIN

In studying cysts of the parenchymatous organs I have been struck by their close resemblance to cystic tumors in other situations, the genesis of which is fairly well established and the life history of which is pretty thoroughly understood. The analogy of these structures to the congenital cystic disease is so close that a detailed examination of them is imperative. While argument by analogy is a dangerous mental recreation, the axiom that things equal to the same thing are equal to each other is as valid in pathology as it is in mathematics.

Spleen. The most interesting of these corollary conditions is the cystic disease of the spleen, of which there are but a few in the literature. In appearance, in structure of the cyst walls, in the reaction of the surrounding tissue and in the cystic contents, the records read identically like those of the polycystic kidney. The origin of the cysts of the spleen was ascribed by Fink[4] to the lymph-vessels. He quotes Kölliker as authority that the lymphatics of the spleen are divisible into a superficial and a deep set, the former occupying the capsule, the latter the trabeculæ. Wedl,[5] was able to trace the lymphatics from the capsule to the depth of the organ along the trabeculæ. Renggli[6] believed the cysts were due to a dipping down of the peritoneal layer of the spleen at an early embryonal period. He bases his view on the appearance of the cells lining these cysts. Aschoff[7] calls attention to the fact that cells lining lymph-vessels may under certain conditions become cuboidal, and accepts the opinion that these cysts are developed from the lymph system. This is well illustrated by a figure published by Schmidt[8] which I have ventured to have reproduced (Fig. 14). Fink's term "cystic lymphangioma" may therefore be accepted with confidence, since it corresponds both to the anatomic findings and to the opinion expressed in the literature. It would seem, therefore, that cystic disease of the spleen furnishes the connecting link between polycystic disease and the cystic lymphangiomata.

In this connection a case of cavernous

[1] Parker: Am. J. M. Sc., 1899, cxviii, 272. Haarer: Memorabilien iv, 8, 1561. Bockheimer, Diss. Würzburg, 1897.

[2] Compare Hertzler: Treatise on Tumors, Lea & Febiger, 1912, Fig. 107, p. 263.

[3] Deutsche med. Wchnschr., 1899, xxv, 677.

[4] Ztschr. f. Heilk., 1885, vi, 406.

[5] Wien. Zitzb., lxiv, abt. 1, p. 301.

[6] Diss. Zürich, 1894.

[7] Lubarsch & Ostertag, Ergebnisse, 1895, ii, 456.

[8] Virchow's Arch. f. path. Anat., 1901, clxiv, 50. Taf. ii, Fig. 5

angioma of the spleen reported by Homans[1] is of interest. The spleen and the peritoneum in the region of the transverse colon were the site of an extensive and progressive cavernous angioma. In general morphology, as well as in mode of advancement, it bore a close relation to the polycystic kidneys associated with pararenal lymphangiomata.

Pararenal cysts. The most striking examples of these analogous conditions that have fallen into my hands are the pararenal cysts. A specimen of this condition was reported and figured by Lejars[2] which I have thought worth while to reproduce (Fig. 15). It consists of a reniform tumor made up of multilocular cysts altogether, as the figure shows, like the polycystic kidney. So close was the resemblance to the kidney that the distinguished surgeon believed he was removing a polycystic kidney. He did not discover the error until the post-mortem, when the patient was found to have two kidneys both of which were cystic. The tumors removed at operation contained no renal tissue. It has been my privilege to examine one such specimen removed from a patient possessed of two normal kidneys. This specimen was not so large as the one described by Lejars but it was reniform and made up of numerous cysts. These cysts were in structure identical with the polycystic kidneys. The endothelium in some places was distinctly cuboidal, though for the most part low cuboidal and papillary formation was everywhere marked. The cystic contents likewise was identical with those of polycystic kidneys. This patient also had small peritoneal cysts in the hepatogastric ligament, presenting as it were a link between the pararenal cystomata and the usual cysts of the peritoneum. At the periphery of the cystic mass were clefts lined with flat endothelium which correspond to similar clefts in the spleen, pancreas and kidney as well as in the cystic lymphangiomata of the forearm and seem to give some hint as to the mode of extension of these tumors.

Parallel with the cystis of the spleen are those of the suprarenal gland. Such a case is reported by Sick.[3] The cyst walls are described as being lined with endothelium and their contents as coagulated lymph, characterized by fibers and bands. The cysts were surrounded by a granulation-like tissue in which numerous lymph spaces were in the course of formation.

Cysts of the tendon sheaths. I have in my possession a tumor obtained from the forearm of a boy four years of age which was noted soon after birth. This tumor is formed by numerous cysts varying from a few millimeters in diameter to the size of a hickory nut (Fig. 16). The cystic contents were straw colored and stringy in some parts and distinctly colloidal in others. The cysts were lined for the most part by cuboidal or flat cells which showed but little tendency to form papillary projections within the lumen. The cystic area gradually extended as the child grew until the palm of the hand was involved. In the advancing border of the tumor the cysts were small and in the connective tissue between macroscopic cysts there were clefts filled with thin fluid. The cells of these clefts presented evidence that the tumor extended by the development of new cysts in the periphery of the tumor. Paltauf[4] reports a similar case following traumatism. In this case the soft parts as well as the bone were destroyed by the advancing tumor and amputation became necessary. He states that the cysts were lined with large clear endothelial cells and calls particular attention to the presence of endothelial clefts and cavities in the connective tissue surrounding the cysts.

Cystic lymphangiomata of the tongue. It has been my privilege to examine a number of tongues which were the seat of cystic disease. In the cases which are well marked clinically small cysts appear within the substance of the tongue (Fig. 17). In this situation, as is well known, the disease progresses by acute exacerbations during which the tissue between and beyond the cysts becomes infiltrated and inflamed. With each exacerbation the cysts increase somewhat in size and number. The lymph spaces beyond these cysts lined with perfectly flat

[1] Ann. Surg., Phila., 1897, xxv, 732.
[2] Bull. et. mém. de la Soc. de Chir. de Par., 1907, xxxiii, 274.
[3] Virchow's Arch. f. path. Anat., 1903, clxxii, 445.
[4] Verhandl. d. deutsch. path. Gesellsch., 1907, xi, 255.

endothelium become dilated during these exacerbations and give perhaps some hint as to the mode of extension. The presence of proliferated connective tissue in the kidney presenting the usual changes of interstitial nephritis may have some relation to the processes just described in the tongue. This suggests that similar processes may be responsible for the extension of the cystic disease of the kidney and for the final destruction of the parenchymatous portion of the organ. The clinical evidence of such a process is found in the advent of uræmia, indicating a loss of function in a kidney which has perhaps for many years been capable of serving the individual adequately. The pathological anatomy shows the chronic interstitial changes associated with acute changes as manifest by round-celled infiltration and cloudy swelling. How many such periods of exacerbation the organ may have suffered is impossible to determine. That they may be numerous is indicated by the fact that exacerbations may occur sufficiently severe to bring them to the surgeon. Such kidneys are often possessed of enough reserve capacity to withstand both the effects of the disease and the interference of the surgeon, only to fail at some future period at a renewed exacerbation. In the case of the cystic lymphangiomata of the tongue, these exacerbations and regressions may be readily observed. Here life is not threatened and the progress of the disease may be studied over a period of many years.

Lymphangiomata of the skin. I have recently examined a tumor of the skin resembling in many particulars those of the tongue. The tumor was moderately elevated above the surface and was composed of numerous cysts of varying size and character of contents. Others of this type have been reported and it is of interest to note that they often follow traumatism. These are unquestionably lymphatic in origin.

Cysts of the peritoneum. Cysts resembling those mentioned above in contents and structure are found in various parts of the abdominal cavity. A number have been reported upon the stomach near the scars of stomach ulcers. Various portions of the mesentery and omentum have been the seat of such cysts. Often side by side cysts may be found filled with thin fluid and colloidal material. Similar cysts have been observed involving the thoracic duct.

In some cysts of the peritoneum it is of interest to note that not infrequently cysts are found side by side which represent the various types. These are often associated with the pararenal cysts and present a convenient link between them and the more widely distributed cysts of the peritoneum. In some the lining cells are more in evidence and the cell contents are colloid with fibrin fibrils within them. Beside them may occur cysts lined with perfectly flat cells and with contents incapable of coagulation. Their histological character, therefore, places them midway between the two groups of cysts. Cysts which result from lymph obstruction, as above, and peritoneal scars, many of those in the skin, and the certainly congenital cysts about the neck are of the latter type. Such cysts would appear to be purely retention cysts, while the former type, lined with taller cells and containing colloid and fibrin filaments, represents an active process.

In discussing this problem it is difficult to avoid the controversy as to the nature and distribution of endothelial cells. No matter how anxious one is to defer to the opinion of the embryologists relative to the shortcomings of His, those who work long with the cells belonging to all the situations included by this distinguished anatomist are forced to the conclusion that endothelium is endothelium, no matter from whence the embryologists derive it. Under similar conditions cells in all these situations act much the same and under reaction present all the types seen lining the cysts in polycystic disease. Aschoff particularly has called attention to the capability of endothelial cells to assume protean forms. Arguments as to the origin of cells from their form alone are always subject to error, and especially so as regards endothelial cells, which even in their normal state refuse to stay placed in any classification. The difficulty is still greater when such cells have been subjected to inflamma-

tory or neoplastic influences. That some of the cells lining these cysts resemble cells lining kidney tubules does not permit judgment upon their fellows which bear no such resemblance. In the illustrations, many of the cells are identical in form and structure with endothelial cells found in other situations. The single histological problem of interest here is, Are the flat cells renal cells which have been compressed, or are the so-called cuboidal cells endothelial cells which are in a state of reaction? The preponderance of evidence is that polycystic disease is a neoplastic process. If so, the cells cannot be renal epithelium flattened by pressure. Papillary processes, even to the second order, as is sometimes seen, exclude any suggestion of a purely retentive process. Anyone who has worked much with endothelium will endorse Aschoff's views above quoted and will see in the finer structure of the nuclei the strongest argument of identity.

In order that the polycystic disease of the parenchymatous organs shall develop from the lymph apparatus of these organs the existence of lymphatics must be established. Unfortunately these have been the object of but little research. For the kidney, Stahr[1] has made a careful study. He demonstrated lymph capillaries along the vessels between the tubules and described them as being about 12 microns in diameter. At the conjunction of the cortical and medullary portion they form arches. For injection he used Gerda's method; for the surface vessels Golgi's method was employed.

With the review of evidence before us one cannot resist the conclusion that the polycystic disease of the parenchymatous organs, including those reported in the spleen as cystic lymphangiomata, are morphologically identical. It would be difficult to conceive, if they were derived from the parenchymatous tissue of each of the several organs, how this identity of structure and function (if the character of the cystic contents may be regarded as a criterion of function) could be brought about. This is particularly striking if one includes those of the spleen where no epithelial origin can be hypothecated.

Harmony in structure and function even in pathological states bespeaks a similar or identical origin. The single structure common to all these situations is the lymph-vessels. Added to this, the similarity, even identity, of the cysts in question with lesions of the lymph system presents the strongest possible argument for the theory that the polycystic disease of the parenchymatous organs is lymphatic in origin and is the result of changes in these channels either congenital or acquired.

Ultimate conclusions are not possible where the premises are still under discussion, but the facts fortunately are more stable than the theories, and so long as these harmonize, tentative conclusions may be made with a certain degree of security.

CONCLUSIONS

Polycystic disease in all the parenchymatous organs is morphologically identical.

It resembles closely in structure and development cystic formations known to be derived from lymphatic channels.

Polycystic disease probably develops from the lymph channels of the organs.

[1] Arch f. Anat. u. Entwickelungsgeschichte, 1900, 41.

A STUDY OF THE CANCER SITUATION[1]

By W. P. CARR, M. D., Washington, D. C.

CERTAINLY there is no more vital question in modern medicine than the cancer problem. And if this statement needs confirmation I simply remind you of the great prevalence of cancer and of its rapid increase, out of all proportion to the increase in population, in all civilized countries; and of the immense amount of time and labor that has been expended, and is now being expended, in the attempt to elucidate its nature and cause and find a preventative.

No problem has given greater promise and yielded such meager results.

Except in our manner of operating, and a greater per cent of cures by earlier or more radical removal, we have made little or no advance in the last hundred years.

There is growing, however, a more hopeful view of the results of early and radical operation, and several of the most influential national medical associations have endorsed the plan of teaching the general public the importance of early diagnosis for this purpose, and the importance of regular systematic examinations by a physician during the period of life when cancer is most common. This work is in the hands of a committee of splendid and enthusiastic men and good may result.

One evil, however, I fear will counterbalance some of the good. How many cancer-phobiacs will we produce who will fall into the hands of quacks to be robbed and made miserable; and how many neurasthenics will be given a new dread with which to torture themselves and their physicians?

We may hope that the good will far overbalance the evil; but we are still far from a satisfactory solution ot the problem.

Let us carefully review the few known facts bearing upon the cause of cancer and see if any practical deductions can be made.

We will consider only epitheliomas, and true carcinomas of epithelial type, which constitute the great bulk of the malignant growths of middle adult life, and which originate, possibly, from causes entirely different from the causes of sarcoma and other malignant tumors.

We know that such tumors always originate in some part or organ containing epithelium derived from the epiblast or hypoblast, and that the tumor is a wild growth of epithelial or epithelioid cells.

Some of these cells may be embryonic inclusions, or unused remnants left over in the developing fœtus, like scraps of cloth in a tailor's shop, that have escaped destruction in the manner provided by nature for cells whose period of usefulness is past; but there is no special reason to believe that they are other than normal epithelial cells stirred to abnormal activity by some unknown cause.

Whether they are embryonal remnants or inclusions, or normal constituents of the body, the fact remains that, after resting quiescent or performing normal functions for years, they suddenly begin to proliferate in a wild and harmful way, to invade surrounding tissues, and to wander to distant parts through the lymphatics, there to start secondary growths.

We know that this invasion, like that of all foreign bodies, is opposed in various ways. The chief opposing force to cancer cells is apparently a proliferation of connective tissue around the invaders in an attempt to wall them up in a fibrous capsule.

There is good reason to believe that many incipient cancers *are* thus walled up, squeezed out of existence by the contracting connective tissues, and cured, like many cases of incipient tuberculosis, without the existence of the disease ever being suspected.

And in *most* cases the growth is in this way held in check and localized for a considerable period.

Unfortunately, when the cancer cells are relatively more active a few of them manage

[1] Presidential address read at the Washington Surgical Society, January 17, 1913.

to escape before the wall around them is complete. They are then able to start new groups and to repeat, and continue repeating, this performance.

Thus the tumor grows with a spongelike formation of acini, filled with cells, surrounded by trabecular walls of fibrous tissue.

We know too that the character and rapidity of the growth depends upon the relative activity of the opposing cell armies. When epithelial proliferation is slow or connective tissue growth rapid in proportion, we have small acini with thick walls and a solid schirrous growth of slow development.

And when the converse is true we have large acini, with thin walls, and a soft medullary cancer of rapid growth and more malignant type.

When we are able to determine the cause or causes for this sudden change in a group of cells from normal activity or quiescence to wild proliferation, we will have practically solved the etiology of cancer.

In the light of our present knowledge three explanations seem possible—

1. Destruction or alteration of the normal nerve control of these cells.

2. Intoxication of the cells by bacterial poison, or other poison.

3. Alteration of the food supply to the cells in question.

Let us look into the possibilities in each case.

Normally, the sensory or secretory nerves send filaments into all parts of the skin and mucous membranes, and to all glandular epithelium. These filaments break up into fine tufts and come in contact with each living epithelial cell. This is shown by Golgi's staining method, as well as by the fact that we cannot put the finest needle point into the skin without touching nerves carrying sensation of pain, or temperature sensations.

We are familiar with the trophic effects of nerve lesions, particularly upon mucous membranes.

I have seen inflammation of the palate and fauces following extirpation of the Gasserian ganglion that for a week had all the appearance of a malignant growth.

We know, also, that by stimulating the secretory nerve of a gland we increase the activity of its secreting cells, and that this is not due, as was once supposed, to circulatory changes.

We know that gastric secretion can be influenced by impulses coming from the brain, that savory odors or even thoughts of food will, in the hungry, cause a flow of gastric juice and saliva, and that anger, grief or fear may often completely check these secretions.

All this goes to show that each living epithelial cell is under nerve influence and control.

What should we expect of a cell suddenly deprived of this control?

Naturally it would become an independent organism, and a foreign body in the animal to which it belonged. It would revert to its ancestral type and become an embryonal cell, or practically an amœba, devoting all its energy to assimilation and reproduction.

And in fact this is just what it does.

I quote from an article of my own on malignant tumors of the breast read before the Washington Obstetrical and Gynecological Society, May 19, 1899, as follows:

Dr. Ludvig Hektoen, in the first volume and number of *Progressive Medicine*, has collated some striking observations on the growth of epithelial cells. Ljunggren, he says, found to his surprise that he could preserve carefully sterilized bits of human skin in sterile human ascitic fluid for months and that the cells of the tissue retained their vitality. Successful transplantation was made with pieces kept in such sterile fluid for a month. The transplanted cells not only grew over the raw surface, but penetrated into the granulation tissue beneath, after the manner of a beginning carcinomatous growth.

The abstract of Loeb's observations on epithelial regeneration by Dr. Hektoen is so clear that I copy part of it verbatim:

"From a margin of a tissue defect huge epithelial protoplasmic or plasmodial masses move in a sliding manner over the naked surface, enclosing and dissolving the crusts and other obstacles. Regenerating epithelium readily removes such substances as cartilage when placed in its way. Below the protoplasmic layer epithelial cells wander in

from the margins of the defect and often grow down into the connective tissue, apparently checking the growth of the latter. The process is *closely allied to changes in carcinoma.* At the same time active changes, such as mitoses, occur in the epithelial cells removed some distance from the margins of the wound."

According to Loeb small pieces of epithelium placed in the center of scabs or crusts send out processes in all directions into the crust, the cells acting as separate organisms *independent* of blood supply or *nervous influence.*

These observations of Hektoen, Ljunggren, and Loeb have since been confirmed by numerous observers, and it is clear that the infiltration of cells from a skin graft into the subjacent tissues is prevented, at first by connective tissue proliferation, and later by the epithelial cells forming new nerve connections and settling down into well ordered epithelium.

It would seem quite possible also that complete severance of a nerve connection may not be necessary, but that altered or abnormal influence may be exerted by sick or injured nerves, or by nerves coming from exhausted nerve cells.

It would seem at first sight that the only conditions necessary to produce cancer are a group of epithelial cells severed from nerve connections and a sluggish reaction of the connective tissue. But there are several other considerations that enter into the problem, such as a suitable food supply for the epithelial cells, the presence or absence of certain cytolysins and of altered internal secretions, and of a bar to the formation of new nerve connections.

The remarkable effects upon tissue growth of the altered secretion of the thyroid, the hypophysis, and to a less extent of the suprarenal body and generative glands must not be forgotten as at least possible predisposing factors.

Altered nerve control may be due either to local or general conditions. Exhaustion and even destruction of central nerve cells may come from purely emotional discharges of nerve impulses.

Crile has demonstrated this in laboratory experiments and confirmed it in clinical studies, and has shown that fear and worry, which is a chronic form of fear, are the most powerful agents for rapidly exhausting brain cells. All thought, emotion, or exertion of any kind does this to a certain extent, and also glandular activity; but muscular exertion does not seem to do it with nearly so much rapidity as emotional excitement of a like degree.

Crile has shown clearly the cumulative action of repeated frights in rabbits, and the microscopic appearance of the brain cells when exhausted in this manner; and that while all emotions are accompanied by more or less rapid discharge of nervous impulses, and oxidation of the cell protoplasm, which may be carried to the point of complete disruption of the cell, yet fear seems more potent than anger or defensive emotions. Sexual emotion probably comes second in exhausting power.

As the result of emotional excess, therefore, or of any kind of excessive brain work, we may have enfeebled or altered nerve impulses going to the various epithelial cells of the body. And under such circumstances we must believe that local pressure of inflammatory products would more easily act to further alter these nerve impulses, or completely block them.

The circulation in the blood of toxic substances must aid in producing such a condition.

Alcoholic neuritis may produce either hypersensitiveness of a nerve or anæsthesia, according to the degree of its intensity.

The same is true of tobacco, lead and uræmic poisons, and probably of many autointoxications of which we as yet know little.

It is easy to conceive therefore how contracting scars, bruises, or prolonged local irritation, by infiltrating the tissues, may press upon nerve filaments and more or less completely block them—especially when the central impulses are weak—and thus leave groups of epithelial cells more or less completely without nerve control.

One of the chief predisposing causes of cancer may be the exhaustion of the central nerve cells, aided by autointoxication of some

form, and the chief exciting cause a local injury or prolonged irritation.

Indeed, I think we will see presently that this is not only possible but very probable.

It does not seem necessary, therefore, to introduce a cancer germ as the cause of these growths.

And yet there are some facts pointing in that direction.

Plant tumors greatly resembling cancer have been produced by inoculation with a bacillus which has been obtained in pure culture and invariably produced such a tumor when introduced into a scratch or prick of the bark of a plant. Mouse tumors of a sarcomatous nature have been caused by inoculation.

Cancer has many of the characteristics of a slow infection.

But as yet no germ has been discovered, though an enormous amount of work has been done in that direction. Cancer transplantation or inoculation has never been successful unless we accept as such the reproduction of secondary growths in those already affected, which may have been produced by simple traumatism in a person having the predisposition.

Failure to grow cancer cells in vitro, except in the serum of cancer patients, points to the presence or absence of something in the blood of cancer patients which is an important predisposing cause, and militates against the germ theory, as in that case we would expect to find an immunizing or inhibitory action of the serum upon the germs and cancer cells.

And such action has been claimed by advocates of the use of ascitic fluid of cancer patients by hypodermatic injection as a cancer cure. Cancers have been produced, on the other hand, by long continued and repeated X-ray irrigations under conditions that seem to preclude the possibility of a germ origin.

The most conservative conclusion at present seems to be that, while some cancers may be due to infection, the probability is that more are not; but are caused by a combination of local and general conditions, and that the latter are largely the result of modern civilized methods of living.

Intoxication of the cells by substances absorbed from the alimentary canal, or by altered internal secretions, is a conceivable cause of cancer, but rather of a predisposing than exciting character. In such case we must look to some local condition for the exciting cause and for the determination of the site of the growth.

This, however, is never difficult to find. I think it is now conceded that cancer seldom occurs, if ever, in normal tissues; but always where there has been a local inflammation or long continued irritation.

I took this ground in a paper published in the *Medical News*, Jan. 11, 1896, entitled, "The Nature and Treatment of Mammary Neoplasms."

At that time it did not meet with much favor, but the idea has been gaining ground every year since and Coley[1] gives an exhaustive and conclusive article sustaining this view.

So, even if we accept the theory of cell intoxication, it is not absolutely necessary to assume a cancer germ as the cause of the poisoning.

But whether we believe in embryonic remnants, cell inclusions, the germ theory, or any other known hypothesis, we can not sufficiently account for all the phenomena of cancer growth without assuming a predisposing cause or causes that are of great importance.

We must also concede this condition to be the result of civilization. For cancer is practically unknown among savages of any country and has increased in direct proportion to the degree of modern civilization in all countries.

Let me relate briefly the sequence of events in one of my earlier cases of cancer of the breast as I interpreted them with the assistance of Dr. Walter Reed, Dr. Jas. Carroll and Dr. Wm. Gray, all now unfortunately dead.

The patient, a woman, received a sharp blow on the breast from the corner of a mantel piece, producing rupture of small vessels and interstitial hæmorrhage acting as a foreign body and producing infiltration of leucocytes and proliferation of connective tissue. Period of quiescence. Contraction of the fibrous tissue. Squeezing of some milk ducts

[1] Tr. South. Surg. & Gynec. Ass., 1910.

and nerve filaments supplying them, making uncontrolled embryonal cells of the epithelial lining of these ducts. Proliferation of these cells. Distention and finally rupture of the thin walls of the ducts. Infiltration of the surrounding tissues with cells — cancer cells. There was no abrasion of the skin in this case, such as seems necessary in the inoculation of plant cancers, and no apparent way for local infection. The tumor was removed in an early stage, and examined microscopically, and proved to be adenocarcinoma. This lady had the predisposing causes of civilized life well marked.

I have studied a number of similar cases with the assistance of the gentlemen just mentioned and, later, with the help of Dr. J. S. Neate, who, sad to say, is also now dead. And I have a few excellent microphotographs made by Dr. Gray, showing some of these conditions.

I think there is reason to believe that some cancers occur in just this way, and that some are squeezed out of existence in the early stages by active or even excessive fibrous growth. This fibrous growth may remain stationary for years, constituting practically an adenoma.

Bruises are exceedingly common. Why are cancers not even more common? Why are they so rare in savages who are more exposed to bruises than civilized people? And why are they so much more frequent about the age of the menopause? Again we are forced to seek the explanation in a predisposing cause or causes — evidently a factor of great importance, most potent about the age of forty-five and not present in savages.

If we can determine this predisposing condition we may accomplish much; for it seems quite as necessary as the exciting cause in a majority of cases.

This leads us to inquire what are the essential differences in savage and civilized life. Three important differences are apparent: (1) The savage gets more fresh air and outdoor life. (2) The savage eats irregularly of simpler food, is subject to long fasting periods, and, on the whole, eats much less *in the proportion to the exercise he takes* than civilized man. He also drinks less of alcoholic beverages, not from choice, but necessity. In consequence of these two things the savage has less toxic material in his blood and body juices. (3) But there is a third and even more striking difference. The savage uses his muscles much and his brain little.

All his expenditure of energy, or nearly all, is muscular, and we have seen that this does not exhaust the central nerve cells like emotional discharges, and intellectual work. He is supposed to have little or no fear; he does not worry over the price of his wife's bonnet; he troubles himself little and for short periods only about the sentimental side of life—he is too busy with the necessities. He knows nothing of jaded nerves and sleepless nights. With modern civilized men and women the exact converse is true. Outdoor life and muscular exercise are at a minimum, and the intellectual and emotional faculties worked to the limit. With civilized man fear is constant. Fear of failure, financial, social, or scientific; and worry, which is little more than a chronic cumulation of little fears, he has with him always. And we have seen how fear is the most *potent* cause of nerve exhaustion.

But he also works his brain hard in business and intellectual pursuits. His wife exhausts hers over household and social matters; and both are constantly using the most rapid means at their command to exhaust, through the emotions, the energy and substance of their brain cells. Often it is not so much the work he does that wears out a man as his misguided efforts at relaxation and the having of a good time after working hours. And the same thing is true in a little different way of our women.

That modern life in all circles of society is more strenuous than ever before can hardly be doubted, and this leads to nervous and digestive disturbances that are of many kinds and varieties, and not infrequently to a peculiar condition which I have come to regard as an almost necessary factor in the production of cancer.

This predisposing cause or precancerous condition is characterized by soft swollen mucous membranes, spongy receding gums, loose lower incisor teeth with accumulation of tartar, flabby tongue, slight chronic bronchitis, more or less thickening and brittleness of the nails, and usually by alternating diarrhœa and constipation. In some there is marked

diminution of the pain sense, as well as of the temperature sense, or there may be alternating hyper and hyposensitiveness. In women the cervix is often flabby, and of a peculiar pale yellow color.

Any excess, whether emotional or alcoholic, or of overeating, or loss of sleep, may bring on exacerbations of any of these conditions, and not infrequently produces abdominal pain or a watery diarrhœa that comes on at a definite period after the excess.

The urine usually contains indican and there may be other symptoms of autointoxication. The underlying cause of this condition I believe is exhaustion of the protoplasm of cells of the central nervous system, similar to that of old age, and yet occurring while the general nutrition of the body and its cell growth is unimpaired.

In this respect the precancerous state is unlike that of old age where all cell activity is lowered, and epithelial proliferation diminished in proportion.

This precancerous state is not clearly marked in every case; but I have seen it so often in connection with cancer that I cannot escape the belief that it is a predisposing cause of great importance, and that many a patient has developed cancer from some local lesion that would otherwise have given no trouble because of the comparative *age of his central nerve cells* and the comparative *youth of his epithelial cells*.

Sometimes only a few of the symptoms may be apparent, and sometimes prolonged local irritation may cause cancer with little or no predisposing cause. Some cases may occur from infection with a cancer germ, with a predisposing cause in some abnormality of the blood. But most cases present evidenc of nerve exhaustion, and also of autointoxication, and both are due to modern methods of living.

While writing this article two women have come to me with cancer of the breast. The first remark of one of them was, "Doctor, I have a knot in my breast that is growing and paining me, and I am all run down from the worry I have had during the past two years."

The other one had well marked Rigg's disease.

I think I have never seen a cancer of the mouth or lips not accompanied by marked disease of the gums.

In view of these facts it seems important that we should recognize this predisposing condition and try to prevent or remedy it before cancer develops.

It is a hopeless task at present to try to make the American people live as they should; but in individual cases we may accomplish much. It is hard to make even invididuals give up their pet excesses, and harder still to make them eliminate fears and worries.

But we can sometimes instill into them the spirit and philosophy of Marcus Aurelius, and modify their extravagant use of nerve cell protoplasm; or send them on a pleasant vacation now and then, and give their cells a chance to recuperate and rejuvenate to some extent. And we can usually improve conditions causing autointoxication and get more muscular exercise and less brain work in their lives.

But the treatment must be suited to each individual case, and will suggest itself to every competent physician. My plea at this time is for *recognition* of a very common condition that I believe is not only an important predisposing cause of cancer but also leads to other serious lesions — a condition, too, that is preventable, and curable, if properly treated before too serious organic changes have occurred in the brain, kidneys and other organs. This condition is primarily and essentially exhaustion of the brain cells or premature aging of the brain cells while the epithelium is still comparatively young and vigorous.

DEPARTMENT OF TECHNIQUE

INTESTINAL ANASTOMOSIS; WITH A REPORT ON THE ASEPTIC BASTING STITCH METHOD

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THE *sine qua non* of intestinal anastomosis is firm approximation of the serous coats of the sections of bowel to be united. When Lembert, in 1820, demonstrated this truth, intestinal surgery became possible. Since then there have been developed innumerable methods and stitches to accomplish intestinal union.

In the early days of intestinal surgery it was considered preferable to make a lateral anastomosis. Stenosis from stricture was feared in the end-to-end method. As this fear was gradually found to be groundless, the use of bobbins and internal splints for assisting end-to-end suture came into vogue. They, in turn, have been given up and to-day the method used by most surgeons is the simple suture of the open ends of the bowel.

Although the open method of anastomosis with the use of rubber-covered clamps is well established as the operation of choice by many surgeons, the evident break in the aseptic technique when the lumen of the bowel is exposed is a real danger. We are not justified in introducing this complication into all intestinal operations if it can be shown to be unnecessary. Given a clean abdomen and rigid preparation for operation, an intestinal resection is not possible without some soiling from the bacteria-swarming lumen in the open method. Although the wonderful tolerance of the peritoneum to infection usually overcomes this defect, still the danger of infection is ever present and we cannot always rely on the possibly lowered resistance of our patient to make up for defects in our technique. It was with the hope of overcoming this defect in the open operation that Dr. Edward Mason Parker and myself devised what we have called the "basting-stitch method." "It may be defined as an easy and rapid method of suturing, applicable to any form of intestinal anastomosis, whether circular, lateral, or end-to-side, whereby the immediate formation of a patent stoma may be accomplished without operative opening of the intestinal lumen and without the introduction into it of any instrument or ligature."

Since the publication by Dr. Parker and myself of our experimental work on animals,[1] we have had considerable experience with the procedure in the human being. It is our desire to call attention again to the advantages of this method, not only from a theoretical point of view but now as a practical and proved procedure. Although the number of cases is not large, they are sufficient to justify our early claims. All the usual intestinal anastomoses are included in the list, as well as pylorectomy and gastro-enterostomy. The stomach cases are recent, and from our limited experience we realize that certain modifications to insure control of hæmorrhage must be developed before we can recommend the method for use on the stomach. Mr. B. G. A. Moynihan of Leeds, England, in his book on duodenal ulcer, has adopted the basting-stitch method of closing the duodenal stump in pylorectomy.

In all cases the method has proved itself perfect, there never having occurred leakage or stenosis. The writer has used it in every case of resection of bowel since 1908 and for the past three years all such operations performed at the Freedman's Hospital, whether by the writer or other surgeons of the staff, have been done by this method.

To quote from the publication referred to above: "The steps of the operation and the advantages which we believe it to possess may be briefly summarized as follows:

"Division of the intestinal walls is made between two narrow-bladed crushing clamps, placed first in actual contact and then slightly separated so that there is left between them a narrow crushed area, consisting practically only of serosa and fibrous coat, which may be divided by the knife or cautery.

[1] Johns Hopkins Hosp. Bull., 1908, May.

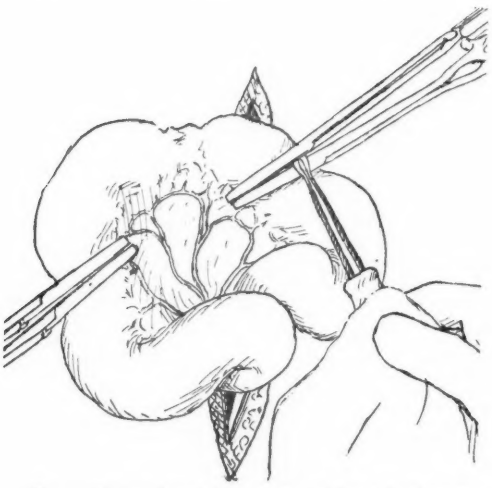


Fig. 1. Clamps (2 pairs) applied on either side of portion of intestine to be resected. Division of intestine with cautery between each pair of clamps.

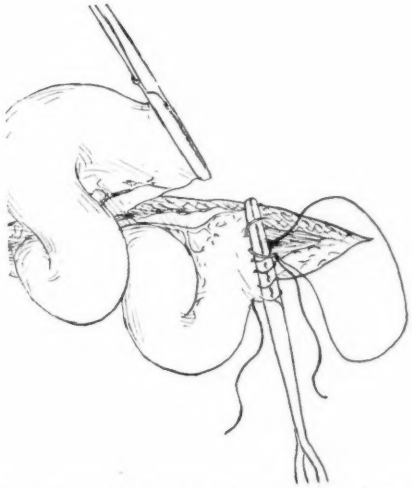


Fig. 2. Resection complete leaving ends of intestine closed by clamps. Basting stitch being applied over one clamp.

"The basting stitch is a Cushing continuous stitch without knots, placed upon the clamped incision with the loops between the stitches crossing over the blades of the clamp. The first bite of the suture applied to either the free or mesenteric border is placed parallel to the axis of the gut; the intervening ones are parallel to the clamp and across the axis of the gut. When the clamp is removed and the stitch at the same time drawn tight, the edges of the incision are automatically inverted and held firmly pressed together in a straight line without any separation of the lips of the opening having occurred. The two incisions, so prepared, are then placed side by side, and are stitched together around the whole circumference of the intestinal tube or of the new operative opening that is to be established. When the last suture has been placed and tied, the basting stitches are cut short and withdrawn, the intestinal canal, or the new anastomotic stoma, then instantly becoming patent. This completes the operation as far as the suture of the intestines is concerned. The lumen has not been opened and it has not been entered by any instrument or other object. The stoma becomes open only with the withdrawal of the basting threads. The steps of the operation are essentially the same whether circular, lateral, or end-to-side anastomosis is done.

"The operation so performed calls for no time-consuming maneuvers and no more than ordinary manipulative skill. The basting stitches are as easily and as quickly placed as the purse-string suture used to turn in the stump of the appendix or grasp the Murphy button. Upon removal of the clamp the stitch is at once drawn tight by a single motion, and the incision, securely closed in a straight line, is then ready for suturing without further attention. The turning in of the cut edges which takes place is automatic and never requires the use of an instrument to assist it, as in the case of the purse-string suture.

"With the two basting stitches in place, intestinal suture becomes singularly free from its usual difficulties. Only a single assistant is ever needed, and even he may be readily dispensed with. The parts to be joined are held in the most convenient position and under perfect control, so that the stitching is done with surprising rapidity and ease.

"The troublesome eversion of the mucosa and the vermicular contractions of the muscular coats no longer exist. Approximation of serous surfaces to any desired extent and at a uniform distance from the margins of the incision is assured. The inversion of the cut edges presents a folded wall through which to pass the stitches, making precision in their application, with secure anchorage in the fibous coat, particularly easy.

"Handling of the intestines is reduced to a minimum. The hands of the operator need scarcely touch them, and those of the assistant not at all.

"In end-to-end suturing the small triangular

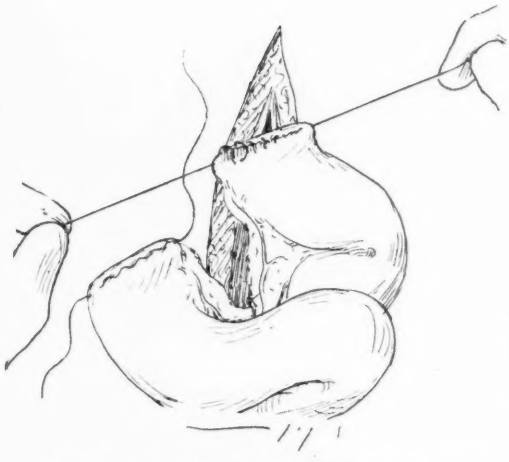


Fig. 3. Clamps withdrawn from under loops of basting stitches and basting stitches drawn taut, automatically invaginating crushed stumps.

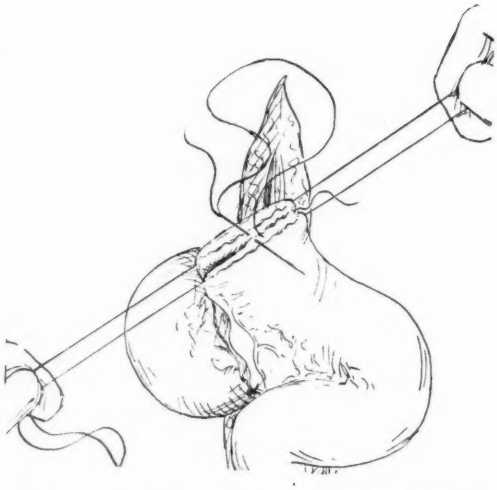


Fig. 4. Inverted stumps of intestine held in apposition on their basting threads. Anastomosing suture begun.

peritoneal defect at the mesenteric border is automatically obliterated by the basting stitch-insuring apposition of serous surfaces at this point.

"A striking advantage of the method is seen when the two incisions to be joined happen to be of unequal length. In this case the longer one is simply puckered or 'gathered' in upon its basting thread until it corresponds in length with the shorter incision and the sutures are then applied. We have successfully united in this way without the slightest difficulty intestinal incisions, one of which was double the length of the other."

This procedure has not been modified materially, though experience has taught us the importance of some of its steps.

No special clamp is necessary for the crushing of the intestinal walls. The writer prefers the ordinary curved Kelly clamp to the one made for the purpose.

It is an advantage to place the clamps across the intestine at an angle of 45 degrees to the long axis of the bowel. This insures a stoma of twice the area of the one made by a transverse division. Then the amount of tissue to be invaginated and the width of the blades of the crushing clamps is of no moment, as the occurrence of obstruction from the formation of a diaphragm is well-nigh impossible. No angulation of the gut occurs after such anastomosis.

Pagenstecher thread is advised for the basting-stitches and can be most easily applied on a curved French needle.

One anastomosing suture of intestinal silk has always been found ample, even in pylorectomy or gastro-enterostomy.

The only accidents that have occurred in our experience have been from not observing the above-mentioned precautions. In one case the basting-stitch (light silk in this instance) broke while being withdrawn. The gut was inverted through the anastomosis, proving the stoma to be patent, and nature was left to pull out the balance of the silk thread. As there are no knots in the basting threads, it was probably dragged into the lumen of the bowel and discharged promptly. Suffice to say that no symptoms were produced and the convalescence was perfect. But since then we have used Pagenstecher linen thread.

In one case of strangulated femoral hernia, done under cocaine anæsthesia, where about seven inches of small bowel had to be resected for gangrene, the abdomen had to be opened on the third day to establish the stoma. Through overconfidence we had failed to break up the adhesion between the bowel walls caused by the crushing clamps. On the patient showing visible peristalsis three days later, the abdomen was opened and the anastomosis delivered through the wound. The intestine was slightly dilated above the line of suture and collapsed below. Union appeared to be perfect. The lateral wall of the distended

loop was invaginated into the anastomosis, effectually breaking up the adhesion between the crushed bowel walls. The stoma immediately became patent and gas was seen to pass into the collapsed bowel. Nothing further was necessary and the anastomosis was returned and the abdomen closed. The patient made an uninterrupted recovery. This case taught us that the clamps put on tight enough to crush the bowel walls and to stop all hæmorrhage also caused an agglutination between those walls. It is necessary to break up this adhesion, by rolling the suture line between the fingers after the basting threads have been withdrawn.

In the operations on the stomach the method worked perfectly for pylorectomy, but the only gastro-enterostomy that we have done showed some hæmorrhage the night of operation. It was not sufficient to affect the patient's convalescence, which proceeded normally without interference other than a stomach lavage. In pylorectomy the clamps are placed transversely across the duodenum and stomach and do not crush across any large vessels. In gastro-enterostomy it is probable that some of the branches of the gastro-epiploic arteries are divided, and that the crushing and cauterizing is not sufficient to control all bleeding. However, it has been suggested that the Connell suture, passing entirely through the stomach wall, be used in making the anastomosis, thus insuring the grasping of any large vessels in the bite of the suture.

Dr. W. A. Jack of Washington has used this method for all intestinal resections and about 1909 adapted it to amputation of the vermiform appendix. The basting stitch takes the place of the usual purse-string suture, and ligation and invagination of the appendix stump is unnecessary. Willard Bartlett of St. Louis suggests the same procedure,[1] being apparently unaware of its use for intestinal anastomosis.

The following is a list of cases of clean aseptic resections and anastomoses done by the writer and some of his colleagues in Washington. There has been no attempt made to gather cases that may have been operated upon in other cities.

CASE 1. Case of thrombosis of the mesentery of ileum close to the cæcum. Resection of 18 inches of affected gut; end-to-side anastomosis of ileum into cæcum. Uninterrupted recovery.

CASE 2. Case of carcinoma of cæcum. Resection of cæcum and ascending colon with end-to-end anastomosis between ileum and stump of colon. After insertion of the basting stitches the stump of the ileum was anastomosed to one end of the closed cæcal stump. The basting stitch was not withdrawn from the balance of the cæcal stump, but was used to permanently close this incision. (The same procedure was followed in pylorectomy.) Operative recovery good. Death two years later of some pulmonary complaint.

[1] Surg., Gynec. & Obst., 1913, Jan.

Fig. 5. Anastomosing suture complete. Gap in mesentery closed. The basting threads being withdrawn. Intestinal lumen then becomes patent.

CASE 3. Case of strangulated hernia in desperate condition. Strangulation of four days' duration with gangrene of bowel. Resection with end-to-end anastomosis. Death. At autopsy no peritonitis and the anastomosis could be demonstrated to hold water.

CASE 4. Another case of strangulated hernia brought to operation too late. This patient died on the second day and at autopsy the same conditions were found as in the previous case. Both of these cases died from obstruction and not from the resection.

CASE 5. Intestinal obstruction in a negro 65 years old. Patient in bad condition. Cause of obstruction not stated. End-to-end anastomosis done after resection. Result, death from obstruction. Anastomosis satisfactory.

CASE 6. Volvulus of jejunum. Resection. End-to-end anastomosis. Recovery.

CASE 7. Volvulus of four days' duration. Resection. Death in forty-eight hours.

CASE 8. Strangulated hernia with undescended testicle. End-to-end anastomosis. Recovery.

CASE 9. Intestinal obstruction from mesenteric thrombosis. Intestinal resection. Death in 12 hours after operation.

CASE 10. Volvulus from adhesion of the bowel to old uterine stump. Resection. Death ten hours after operation.

CASE 11. Carcinoma of cæcum. Resection with end-to-end anastomosis. Recovery.

CASE 12. Case of ileus from band of seven days' duration. Resection. Death second day after operation. No peritonitis. Bowels had moved before death.

CASE 13. Obstruction (late) of lower end of ileum. Resection with end-to-end anastomosis. Death. Autopsy showed no peritonitis and anastomosis patent.

CASE 14. Carcinoma of sigmoid. Resection with end-to-end suture. Death.

CASE 15. Strangulated hernia with gangrene of bowel. Resection. Living 14 days after operation.

CASE 16. Resection of Meckel's diverticulum. Recovery.

CASE 17. Strangulated hernia with gangrene of bowel. Resection of 14 inches of small intestine. Recovery.

CASE 18. Resection of Meckel's diverticulum. Recovery.

CASE 19. Acute obstruction from bands. End-to-end anastomosis. Recovery.

CASE 20. Strangulated hernia with gangrene. End-to-end anastomosis. Recovery.

CASE 21. Volvulus. Resection with end-to-end anastomosis. Recovery.

CASE 22. Strangulated femoral hernia. Resection with end-to-end anastomosis. Signs of beginning obstruction on third day. Abdomen opened and stoma established by invagination. Recovery.

CASE 23. Pylorectomy for pyloric stenosis from indurated ulcer. Recovery. Eighteen months later, gastro-enterostomy for cancer in region of anastomosis.

CASE 24. Gastro-enterostomy for ulcer of duodenum. Hæmorrhage night after operation. Recovery.

CASE 25. Obstruction from Hirschsprung's disease. Side-to-side anastomosis between ileum and sigmoid. Recovery.

CASE 26. Same case. Resection of hypertrophied colon. Death several weeks after operation, apparently from inanition.

All of the preceding cases were resections or anastomoses in clean cases, i. e. where there was no peritonitis. Although the mortality is high, in no case were there any symptoms of peritonitis or stenosis nor did autopsies reveal either complication. The cases that were lost were obstruction cases brought to hospital too late to save by any means. All the fatal cases had been obstructed at least four days and one seven.

In addition to the above, the basting stitch method has been used to perform anastomoses after resection in the following cases. Here it was used, not on account of the fact that it is an aseptic method, but because in the opinion of the operators it is the most rapid and easiest method to perform.

CASE 1. Gangrene of ileum which had been pulled into a perforated uterus. Resection of three feet of ileum. Operative recovery. Death three weeks later from starvation.

CASE 2. Stab wound of abdomen. Resection of injured intestine with end-to-end suture. Recovery.

CASE 3. Tear of transverse colon in case of perforation of uterus. End-to-end suture after resection. Recovery.

CASE 4. Gun-shot wound of abdomen. Resection and end-to-end anastomosis. Death in a few hours after operation.

CASE 5. Gun-shot wound of small intestine. Immediate operation. Resection. Recovery.

We submit that the basting stitch method of intestinal anastomosis is rapid, simple, safe and aseptic.

I wish to thank Drs. E. M. Parker, S. D. Breckinridge, E. A. Balloch, W. A. Jack and S. L. Carson for the privilege of reporting their cases of resection by the basting stitch method.

REFERENCES

1. Johns Hopkins Hosp. Bull., 1908, May.
2. J. Am. M. Ass., 1909. lii, 1107.
3. Surg., Gynec. & Obst., 1912, xv, 504.

HARELIP, WITH ILLUSTRATIVE CASES

BY RAYMOND C. TURCK, M. D., JACKSONVILLE, FLORIDA

INCOMPLETE or simple uncomplicated harelip in children is not a dangerous condition and is of no especial importance beyond its cosmetic aspect.

Complete harelip, however, whether single or bilateral, is almost always complicated by cleft palate and in the double form by a protruding premaxilla. The combination always impedes or entirely prevents breast or bottle nursing. A moderate palatal defect is in itself often sufficient to prevent the child from forming the oral vacuum necessary to suckling. Spoon-fed children are almost always marasmic or in varying degrees of malnutrition and the mortality of the older children is increased because food lodges in the nasal cavity, decomposes and subjects them to digestive disturbances, as well as to bronchopneumonia, and frequently tends to the formation of excessive adenoid and other benign or malignant tumors.

While breast or bottle feeding is never entirely satisfactory in any child with a cleft palate, yet by providing a sound lip and by using a bottle with a large nipple the child may be carried along to the time of greatest safety in operative repair of the palatal defect.

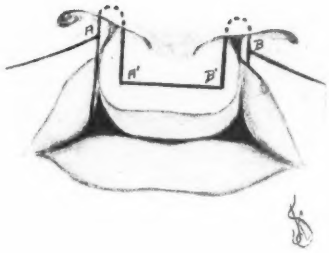


Fig. 1.

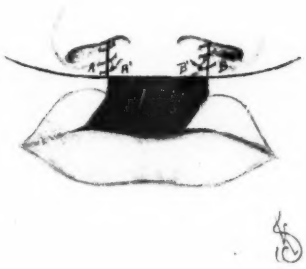


Fig. 2.

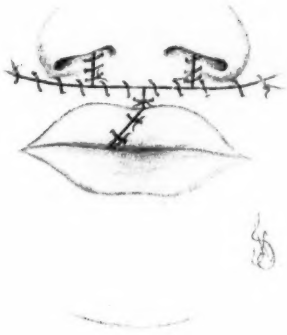


Fig. 3.

In addition to the positive dangers, defective phonation should be considered. Once the child learns to talk with a palatal defect it is almost impossible ever to gain perfect speech, defective pronunciation of consonants persisting throughout life.

In considering the best time for operation we must therefore take into account three factors:

First, the actual operative danger.

Second, the dangers arising from the untreated condition.

Third, the effect upon the child's speech.

The operative mortality in harelip is practically nothing if the operation is properly done, the anæsthetic carefully administered, and excessive hæmorrhage prevented; danger is added when the premaxilla is attacked; any work upon the palate is a serious matter and carries with it a relatively high operative mortality.

Volumes have been written as to the best and safest time for the repair of harelip and cleft palate. Followers of Wolff, Brophy and Lane in their advocacy of palatal repair in the first weeks of life are quite as enthusiastic as the adherents of Kirmisson and others who delay operation until the fourth year.

The writer has unfortunately had a number of late cases, not from choice but because of parental neglect or fear of operation, and this is a factor that the surgeon cannot always govern. In selective cases, however, the writer has adhered to the following plan which in severe complicated cases has worked out very satisfactorily, even though it reverses the usual order of procedure

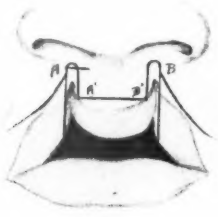


Fig. 4.

Fig. 5.

Fig. 6. Fig. 7.

Fig. 6. G. D. age 16. Bilateral incomplete harelip, detached premaxilla, central cleft palate.

Fig. 7. G. D. Twelve days after author's modification of the Maas-von Langenbeck operation.

in that the lip is repaired first so that the child may by provided with some power of nursing and thus become strong enough to withstand the more severe palatal repair.

A. The work upon the lip and alveolar process is done between the 3rd and 6th weeks by one of two methods:

First. In moderate cases the premaxilla is replaced by the Ferguson method; the lip is repaired at the same time by the operation indicated in the individual case.

Fig. 8. Fig. 9.

Fig. 8. L. H., age 15. Double symmetrical cleft palate and harelip.

Fig. 9. L. H. Photo taken tenth day after first operation immediately after the last stitches were removed. The resultant correction of the nostrils is well shown. The pits in the lower lip are the openings of ducts from sub-maxillary salivary glands — clear saliva, dripped almost constantly from these ducts.

Fig. 10. Fig. 11.

Fig. 10. L. H. Ten days after first operation. Profile showing protruding lower lip.

Fig. 11. L. H. Two weeks after operation for transplantation of the salivary ducts into the mouth, and cuneiform resection of the lower lip.

Second. In cases of markedly protruding premaxilla the work is divided into two stages: First, replacement of the premaxilla; and second, the lip repair two or three weeks later.

B. Palatal defects are divided into two classes:

First. Narrow single clefts which seem suitable for repair by the Brophy method — these cases are best done in the 2nd or 3rd month.

Second. Extensive clefts in which some form of flap transference is necessary. This work is done in the latter part of the second year. At this time the child is usually in good strength and has not yet learned to talk; the results therefore so far as phonation is concerned are all that may be expected, and the operative danger appears to be no greater at the age of two than at four.

There are three general principles of successful plastic work:

1. Broad clean denudation, with no scraps of skin or mucosa left to be closed in the sutured line.

2. Accurate approximation, with perfect hæmostasis, yet with assurance of a plentiful blood supply and care that the sutures are not tied so snugly as to produce a tissue necrosis from pressure.

3. Approximation without tension. This is probably the most difficult problem. The tendency is to make the tension relieving incisions neither long enough nor deep enough. Wide, deep incisions, with tension sutures properly placed and secured will usually provide against undue tension on the coaptation stitches.

The cases illustrated herein have been selected from patients treated under the auspices of The Cummer Fund for Ruptured and Crippled Children at St. Luke's and at Brewster hospitals.

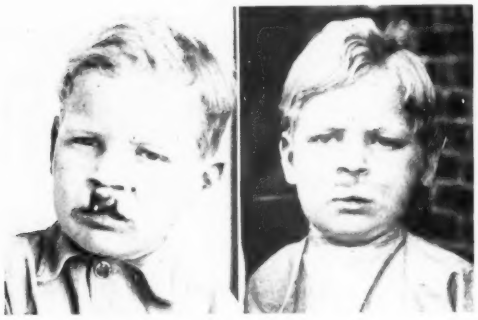


Fig. 12. Fig. 13.

Fig. 12. D. B., age 4. Double complicated harelip, detached premaxilla and cleft palate.

Fig. 13. D. B. Two weeks after final operation. Repair of lip by author's modification of the von Langenbeck operation.

None of the photographs have been retouched and the final pictures were all taken almost immediately after the last stitches were removed. The ultimate results therefore have been better than would appear from the photos, since scars of incisions and sutures fade and in some instances practically disappear as time goes on.

The writer has modified the Maas-von Langenbeck operation for double complicated harelip, utilizing when possible the semicircular denudation of Malgaigne in the upper angles of the defect, and using the flap of the Mirault operation to prevent notching in the vermillion border. The cosmetic results are very good, as may be noted in the accompanying illustrations.

Ether is the anæsthetic used. After the child is asleep it is placed in the Rose position and the anæsthesia continued with the Rupert warm ether vapor apparatus, using ordinarily the silver nasal tube. Hæmorrhage is controlled by pressure of the second assistant's fingers on the facial arteries as they ascend the lower jaw, or by clamps at the angles of the mouth. The field is prepared by painting externally with 3½ per cent Tr. iodine and by sponging with 60 per cent alcohol in the mouth and nostrils.

The median lobule is denuded squarely as in the von Langenbeck operation; the outer edges of the defect are denuded as shown in Fig. 1. The denudations in the upper angles of the defect are made by the semicircular incisions of Malgaigne. This assures accurate approximation in a straight line after swinging the nasal alæ inward and downward. Mirault's denudation of one side of the vermillion border, with flap formation on the other side, is used if the vermillion

Fig. 14. Fig. 15.

Fig. 14. B. H., age 3 months. Double complicated harelip and cleft palate. Ferguson operation on premaxilla at 3 months. Turck's operation on lip at 4 months. Von Langenbeck's operation on palate at 10 months.

Fig. 15. B. H., at age 11 months. Final result.

border extends upward beyond the lower edge of the median lobe, as in the case of L. H., Fig. 8. Points A and B, Fig. 1, are then selected so that the width of the lateral flaps which are to be brought together in the median line of the lip, when added to the denuded median lobule, will a little more than equal the desired vertical lip length.

The outer segments of the lip are then freed from the maxilla if necessary, and the alæ of the nose loosened, taking care to hug the bone and thus prevent excessive bleeding. The points A and B are then drawn to the angles A′ and B′ and the outer segments sutured to the median lobulæ as shown in Fig. 2. This procedure usually satisfactorily narrows the nose and restores the normal contour of the nostrils.

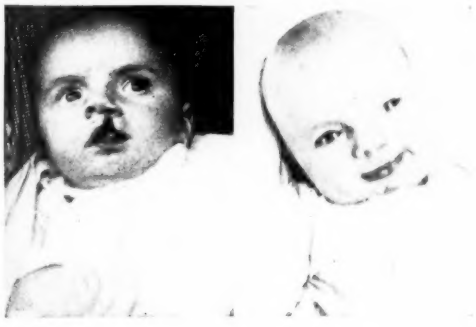


Fig. 16. Fig. 17.

Fig. 16. B. S., age 3 months. Incomplete, unilateral harelip with flattened nostril.

Fig. 17. B. S. Three weeks after correction of defect in lip and restoration of nostril.

Lateral liberating incisions are then made outward from the points A and B and the lower flaps carried to the median line and sutured as shown in Fig. 3. Alternate sutures of silkworm gut and horsehair are ordinarily used, the former passing through and through the lip, the latter through skin only.

In cases of incomplete bilateral harelip, as G. D., Figs. 6 and 7, the same general denudation is made except that the Mirault flap is not used, the outer incisions being carried directly downward and slightly outward as shown in Fig. 4. Notching is provided against by making the lateral relieving incisions somewhat curved, with the greatest width at the median line as shown in Figs. 4 and 5.

The following case is reported in detail as interesting in that he presented an unusual anatomic anomaly.

L. H., a colored male, age 15, was referred to the Cummer Fund for Ruptured and Crippled Children by Dr. Graham E. Henson, and admitted to Brewster Hospital, Jacksonville, in January, 1912, with double complicated harelip, detached but not protuberant premaxilla, cleft palate, flattened nostrils and ectropion of lower lip (Fig. 8).

Ducts of the submaxillary salivary glands opened in the vermillion border of the lower lip. The location and appearance of the openings is well shown in Fig. 9. Clear saliva dripped almost constantly from these ducts, the amount increasing upon any excitation of the olfactory or gustatory nerves. A probe could be passed in each duct for one and three quarter inches downward and outward toward the submaxillary glands.

First operation. Brewster Hospital, February 1, 1912; ether anæsthesia in Rose position. The borders of the clefts in the alveolar process were denuded and the premaxilla was anchored with one silkworm-gut suture. The upper lip was repaired by the writer's modification of the Maas-von Langenbeck operation. The suturing was done with alternate deep, or through and through, silkworm-gut stitches and superficial, or skin, stitches of horsehair. A tension suture of silkworm gut secured at each end by a large split buckshot was inserted. The pits made by pressure of the buckshot may be observed on each cheek in Figs. 9 and 10. These pits disappeared entirely as may be observed in Fig. 11. Primary union resulted; all sutures were removed by the tenth day.

Second operation. Brewster Hospital, February 14, 1912; ether anæsthesia. The salivary ducts were dissected out, the openings were preserved intact, together with a ring of surrounding mucosa, and were transplanted to the oral cavity. The mucosa surrounding the opening of each duct was sewed to the split mucosa of the cheek with catgut, just below and external to the angle of the mouth on the corresponding side.

The protruding lower lip (Fig. 10) was then resected by cuneiform incision and the skin united to the oral mucosa with deep silkworm gut, and superficial horsehair sutures. Primary union resulted. The salivary ducts functionated perfectly in their new location. The final result is shown in Fig. 11.

THE MODERN EXTRAPERITONEAL CÆSAREAN SECTION WITH A DESCRIPTION OF THE BEST TECHNIQUE FOR ITS PERFORMANCE[1]

By BARTON COOKE HIRST, M. D., Philadelphia

IT is unnecessary, perhaps, to review the history of Cæsarean section before a society whose members have access to one of the best medical libraries in the world, but a brief retrospect makes the present position of the operation more easily understood.

Cæsarean section, prior to the seventeenth century, has no practical interest, for aside from the apocryphal case of Nufer and the supposition that the operation might have been practiced by some primitive tribes and peoples, only the post-mortem section was known. From 1610, the date of Trautmann's operation, it was performed with increasing frequency on the living woman but with a mortality almost incredible. The figures are no doubt known to the members of the Society: a fifty-two per cent mortality for the United States, a period of ninety years in Paris without a single recovery, and so on. It was this frightful mortality that prompted the suggestion of Jörg, Ritgen and Baudelocque to do an extraperitoneal section and that induced Gaillard Thomas of New York and his followers to resort to laparo-elytrotomy with a mortality of 50 per cent. Porro introduced his operation at about the same time, reducing the mortality one half in spite of the crude, archaic treatment of the stump. My first Cæsarean sections were done by transfixing the cervical stump with skewers and tying a rubber tube around it, until our fellow townsman Baer taught us how to deal with the cervical stump after hysterectomy. Sanger six years later sutured the uterine wound securely, still further improving the results of Cæsarean section. There remains, however, cause for dissatisfaction with the operation. It is true that a

Fig. 1. The length and situation of the incision.

mortality of less than one per cent has been achieved in a series of more than one hundred cases with every condition favorable to success, but on the contrary, Routh's recent statistics of almost five hundred cases in Great Britain, in which there was reason to suspect pre-operative infection of the birth canal from futile attempts at delivery, gave a mortality of seventeen per cent. It was to avoid this mortality in suspected cases that Frank again resorted to an extraperitoneal form of operation in 1906 by making a suprasymphyseal section of the lower uterine segment under the anterior reduplication of the peritoneum. The importance of this procedure was immediately recognized in Germany; it was extensively tried by all the prominent operators, and, I am told by Dr. William R. Nicholson, who has made the most extensive study of the subject of anyone in this country, was modified by so many that there are now sixteen different techniques for its performance.

There must be reasons for our failure to recognize the merits of a procedure now in its new form seven years old and approved by many of the best surgeons in the world.

These reasons, I should say, are that there still lingers in our minds a disagreeable recollection of the failure of laparo-elytrotomy here; that the operation is believed to be difficult, tedious and complicated, and that the results have not been as good as were anticipated, the mortality in the German clinics having averaged six per cent.

But an impartial student of the subject must admit, I think, that our prejudice has been unwarranted. If one selects the best technique, the operation is simple and easy. The mortality of six per cent in Germany must be contrasted with the seventeen per cent of Routh's statistics, for it

Fig. 2. Incising the parietal peritoneum.

was for suspected cases that the operation was devised and has been for the most part performed. Moreover, these advantages of the extraperitoneal section must be admitted by any candid, unprejudiced person: case for case, an extraperitoneal operation should give the patient greater security; if the uterine wound is infected

Fig. 3. Incising the visceral peritoneum over the lower uterine segment where it is loose and detachable.

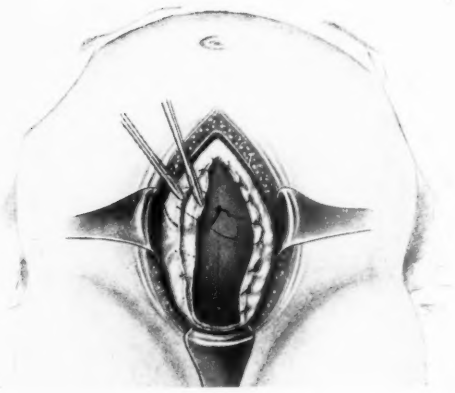


Fig. 4. Uniting the two peritoneal flaps, parietal and visceral, thus closing the peritoneal cavity. The lower uterine segment is then incised and the fœtus extracted. The wound in the uterus is sutured with a two-tier stitch and the peritoneal flaps brought together over it with two or three interrupted stitches.

or gapes after the operation the woman is not necessarily killed; there are no intraperitoneal adhesions subsequent to the operation; the abdominal wound is small and not liable to hernia, and the convalescence is strikingly comfortable and undisturbed compared with that of the intraperitoneal operation.

My experience with extraperitoneal Cæsarean section comprises nine operations in the last eleven months without maternal or fœtal mortality. Two of the patients were presumably infected beforehand and one had placenta prævia.

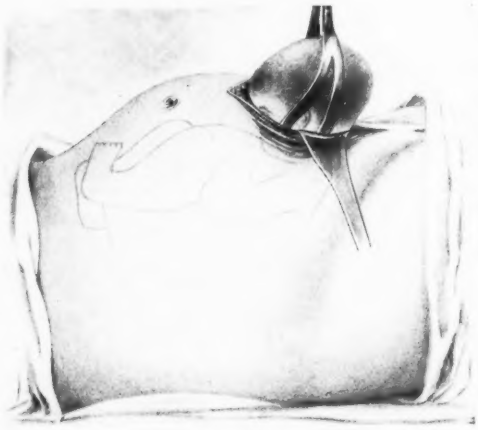


Fig. 5. The extraction of the fœtus with forceps.

Fig. 6. Holding the infant face downward for a few moments to let the liquor amni run out of its mouth and lungs.

The first three operations were done by Frank and Bumm's technique; the next three by Sellheim's; the last three by the Veit and Fromme technique with a slight original modification which decidedly improves the operation, I think.

It consists in suturing the peritoneal flaps before opening the uterus. This technique I shall adhere to in the future, as it is, I believe, the simplest, easiest, and most satisfactory yet devised. The lower abdomen is incised down to the symphysis pubis; the parietal peritoneum is opened in the usual manner; the lower visceral peritoneum on the lower uterine segment is opened in the same way. The two peritoneal flaps are sewed together on each side, thus closing the peritoneal cavity; the lower uterine segment is incised from above downward, as low as necessary to deliver the child, the bladder being protected by a retractor over the symphysis; a forceps is applied to the child's head; the placenta and membranes are removed; the uterine wound is united by a two-tier catgut stitch; the double peritoneal flaps are brought together in the middle line over the uterine wound by two or three interrupted stitches, and the abdominal wound is closed in the usual way.

THE TECHNIQUE OF INSERTION OF INTRATRACHEAL INSUFFLATION TUBES[1]

By CHEVALIER JACKSON, M. D., Pittsburgh, Pennsylvania

THE discovery of Melzer and Auer and the developments by Elsberg, Janeway, Carrel, Quinby, Cotton, Robinson, and others have placed intratracheal insufflation anæsthesia on a firm basis. Each day brings a new class of cases to which it is adapted, so that soon we will all be wondering how we got along without it.

Practically all authorities are now agreed that the larynx should be inspected before the insertion of the insufflation catheter or tube, for the purpose of ascertaining whether or not there is disease present in the larynx, and also to determine the size of the larynx so that the size of the insufflation tube may be selected accordingly, in order to make sure that there is an ample laryngeal lumen around the tube for the return flow. Not only do the sizes of larynges vary in normal individuals, but the laryngeal lumen may be modified by lesions present or past. There is another reason why the larynx should be inspected; namely, throughout the whole category of diseases to which human flesh is heir, it is a frequent thing for patients to date complaints from some particular period, accident, or operation in cases in which the physician or surgeon is absolutely certain that the disease existed long before the incident blamed by the patient. In view of this, it behooves the surgeon to *know* whether the larynx is diseased or not at the time the insufflation catheter is inserted. One such case has occurred where the patient dated laryngeal trouble from the taking of an anæsthetic given by the ordinary open method. The author's own case records and sketches showed that the larynx had been the seat of an infiltration of tuberculous origin four years before the anæsthetic was given, showing that the anæsthesia was in no way responsible, and showing, also, the necessity of knowing the state of the larynx beforehand. Only one thing seems to deter anyone from using the method advocated by Elsberg of inspecting the larynx and passing the catheter or tube by sight. This is the lack of confidence in the ability promptly and skillfully to expose the larynx with the laryngeal speculum. No one capable of giving an anæsthetic should hesitate for one moment about this procedure, if he will take the trouble to pay attention to a few points. These points are:

1. The patient should be fully under the anæsthetic by the open method so as to get full relaxation of the muscles of the neck.

2. The patient's head must be in full extension with the vertex firmly pushed down toward the feet of the patient, so as to throw the neck upward and bring the occiput down as close as possible beneath the cervical vertebra.

3. No gag should be used, because the patient should be sufficiently anæsthetized not to need a gag, and because wide gagging defeats the exposure of the larynx by jamming down the mandible.

4. The epiglottis must be identified before it is passed.

5. The speculum must pass sufficiently far below the tip of the epiglottis so that the latter will not slip.

6. Too deep insertion must be avoided, as in this case the speculum goes posterior to the cricoid, and the cricoid is lifted, exposing the mouth of the œsophagus, which is bewildering until sufficient education of the eye enables the operator to recognize the landmarks.

The most important thing of all is the position of the patient, and next to that comes recognition of the epiglottis, and next the proper motion of lifting the hyoid bone to expose the larynx.

The correct position will be understood by reference to the illustrations. In Fig. 1, the patient is placed on a pillow in a natural position. The larynx can readily be examined in this position, if it is desired merely to inspect it, and is useful for laryngeal diagnosis and some endolaryngeal operations (Johnston), especially in certain conditions of the spine where it is undesirable to extend the head; but for the insertion of an insufflation tube, bronchoscope, or other instrument, it is absolutely necessary for any but the most expert to have the head in full extension. It has been customary to draw the head over the table to gain the full extension in the Boyce position, and for bronchoscopy this is needed for the purpose of moving the bronchoscope about so as to enter the right or the left lung as desired. But for the insertion of the insufflation tube, it is quite unnecessary to have the head extend beyond the table, and in fact it is undesirable. Some years ago I devised the "elbow rest position," so called because the operator's left elbow could be rested upon the table during long endolaryngeal opera-

[1] Read before the Clinical Congress of Surgeons of North America, New York City, November 14, 1913.

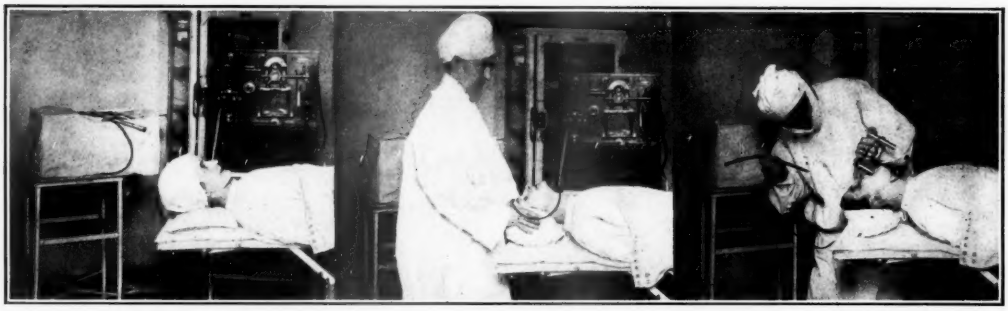


Fig. 1. Photograph of patient with head upon a pillow, the head flexed. In this position it is easy to examine the larynx with the laryngeal speculum for diagnosis, but the larynx will not be exposed in a line with the tracheal axis so that this position is not adapted to the passing of tubes through the speculum.

Fig. 2. The pillow is removed, the head is flat on the table and the anæsthetist is beginning to force the head into the extended position. The thumbs are on the forehead and the fingers are at the side of the head. The direction of motion is shown by the dart.

Fig. 3. The anæsthetist is lifting with the *lip* of the speculum in the direction of the dart. The speculum is always held in the left hand. The right hand, of which the index has been protecting the upper lip, has now received the catheter from the nurse.

tions. For the introduction of insufflation tubes this position of the patient is admirably adapted. All that is necessary to do to the patient is to remove the pillow, place the thumb of each hand (as shown in Fig. 2) on the forehead of the patient with the hands at the sides of the head and then force the forehead vigorously downward and backward, causing an anterior movement of the skull on the atlas and throwing all the cervical vertebra forward. (Upward in the recumbent position.) The effect of this is to throw the hyoid bone and all the tissues of the neck, including the larynx, high up and to elevate the tongue. The neck and shoulders are arched up away from the table. In a fully relaxed patient, it may be necessary for an assistant to steady the head in this position, while the anæsthetist takes the speculum, always in the left hand, his right index being used to pull the upper lip of the patient out of the way so that the lip will not be pinched between the speculum and the upper teeth. The spatular end of the speculum is now inserted posteriorly to the tongue over the dorsum of which it is passed until the epiglottis comes into view.

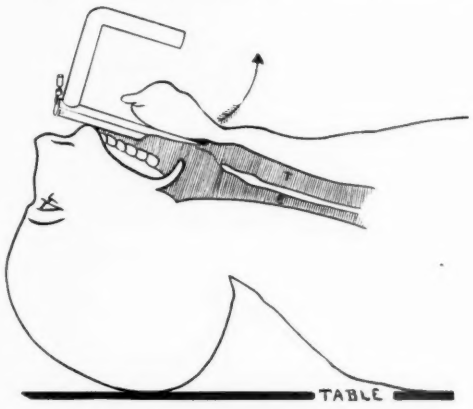


Fig. 4. Schema illustrating the direction of motion to be imparted to the laryngeal speculum in exposure of the larynx for the introduction of ether insufflation tubes.

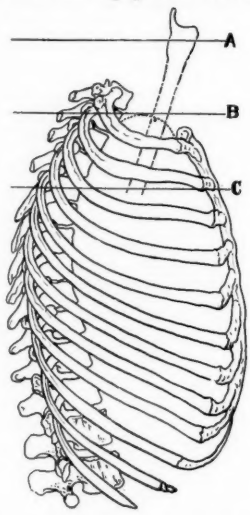


Fig. 5. Schema illustrating approximately the average normal direction of the trachea in the living. The direction is not vertical but backward as well as downward.

The spatular end is made to pass posteriorly to the tip of the epiglottis, and inserting the speculum a distance of, on the average, about half an inch, the hyoid bone and all of the attached tissues are lifted by a motion which is best expressed as the suspension of the head of the patient on the epiglottis by the *tip* of the spatular end of the laryngeal speculum. Great care is necessary at this point not to use the upper teeth as a fulcrum upon which to pry upward with the tip. The motion is rather the lifting of the epiglottis and especially the hyoid bone by the tip of the instrument just as if it were desired to lift the patient's neck upward. Hyoid bone elevation opens the laryngeal door. After the larynx is exposed, the right hand releases the upper lip, which is now safe, and the catheter of the desired size is handed to the anæsthetist by the nurse and the introduction is simple and easy, because the trachea is in a straight line with the laryngeal speculum. This is the great advantage of the extended position with the head on the table. At first sight, it might be thought that the speculum, as shown in Fig. 3, could not be in line with the axis of the trachea. It seems to be the erroneous conception quite prevalent among the profession that the trachea is perpendicular in the neck and chest. As a matter of fact, it enters the chest in a direction backward as well as downward, as illustrated schematically in Fig. 5, so that in the extended position proper for the exposure of the larynx and the insertion of any sort of tube into the trachea, the axis of the speculum, as shown in Fig. 3, is precisely in line. This must be remembered in placing the patient in position, but for the insertion of the speculum it is well to forget it and remember only that the motion is a strong lifting of the tip of the speculum, as shown in the schema, Fig. 4.

Mention is made above of deep anæsthesia. Once the knack is acquired, no anæsthetic whatever, general or local, is needed to expose the larynx in any patient; but to the beginner it simplifies the acquiring of the knack of direct laryngoscopy to abolish the reflexes of vomiturition and coughing, and to abolish entirely the antagonism of certain muscles attached to the hyoid bone. In the author's clinic an anæsthetic, general or local, is never used for direct laryngoscopy, bronchoscopy or œsophagoscopy in any child under 6 years of age, and rarely in adults, except for a few special procedures; but for insufflation anæsthesia, the patient may as well be put completely under by the open method as only partially. To cocainize the larynx for the insertion of an insufflation tube to help along in partial anæsthesia is an utterly needless waste of time.

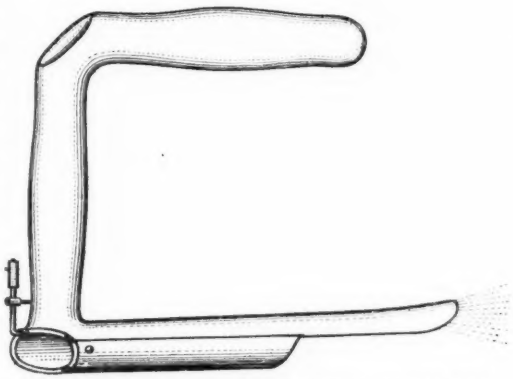


Fig. 6. Laryngoscope or laryngeal speculum with slide opening at side instead of on top. This form, originally devised for other purposes, has advantages for the introduction of insufflation tubes, because the slide may be left off altogether, the insufflation tube being passed out sidewise in the removal of the laryngoscope.

For the introduction of insufflation tubes the side opening laryngoscope (Fig. 6) has some advantages. After the catheter is inserted, the laryngoscope may be removed sidewise through the lateral opening. After skill in direct laryngoscopy is acquired, the slide may be left off entirely, but at first one is apt to be troubled by the tongue curling in and obstructing the view. This is prevented by passing the speculum to the right of the tongue, thus leaving the tongue on the closed side of the speculum.

Incidentally, it may be mentioned that post-anæsthetic laryngoscopy in 80 cases has shown that there is no reaction in the larynx from the presence of the insufflation tube, even in a prolonged anæsthesia by insufflation. In quite a number of the cases anæsthetized by the ordinary open method there has been quite a good deal of reaction, probably from ether mucus bubbling back and forth in the larynx, so that from an observation of these 80 cases the author is prepared to say that there is less irritation of the larynx from an intratracheal insufflation than from an anæsthesia of corresponding duration by the open method.

ILL EFFECTS FROM ILEOSIGMOIDOSTOMY

Report of Case

By AXEL WERELIUS, M. D., Chicago
Surgeon to the South Shore Hospital

THE following case is of some interest in being probably one of the first to be reopened after ileosigmoidostomy, in showing harmful results from improperly placed anastomosis and from the fact that the patient was in complete ignorance that the operation had been done or why it had been performed.

The patient, a female, 28 years of age, housewife, nullipara, was admitted to the South Shore Hospital, January 29th.

Present history. Extends back a year and a half following a supposed uterine operation. Since then she complains of vague, indefinite pains in the lower abdomen, which at times radiate all over the belly. These pains are not influenced by the taking of food, time of the day, nervous excitement, or atmospheric changes, but are somewhat aggravated by exertion. The patient has a sensation as if something was moving to and fro in the belly and feels a slightly tender, movable lump in the lower left abdomen. Following the operation the patient suffers from an irregularly recurrent diarrhœa. Appetite has lately been poor. There is no vomiting, no hæmatemesis, no melena and no jaundice. There are no symptoms referring to the genito-urinary, nervous, respiratory or cardiovascular systems. Mind is normal; special senses perfect. There is some loss of weight.

Past history. The patient was operated on for some uterine affection one and a half years ago. Otherwise the past history is negative. Menstruation is normal. Family history negative. Habits good.

PHYSICAL EXAMINATION

General appearance that of a fairly well-nourished young woman. Head, neck, chesk and extremities negative. Abdomen: On palpation is felt an elongated, somewhat firm, slightly tender tumefaction in the lower left abdomen. The tumor is freely movable.

Blood examination: Erythrocytes 4.200000. Leucocytes 8.500. Hæmoglobin 85 per cent. Blood pressure 125. Urine negative. Examination of the stools was negative.

Diagnosis: Intestinal neoplasm.

Operation: Date, January 31st, under ether anæsthesia. Dr. H. Hueck, assistant, Dr. Rahe, anæsthetist. Incision was made for about four inches through the lower part of the left rectus. On opening the peritoneum there presented a large pouch of bowel, seemingly of the colon. At first I thought that I was dealing with an intestinal anomaly, but on more careful examination I found that the pouch was the lower cut-off end of the ileum that had been anastomosed to the sigmoid. The ileosigmoidostomy had been done at some distance from the cut-off iliac end, thus forming a large intestinal appendix (Fig. 1).

The pouch was about 10 cm. long and 4.5 cm. wide, greatly thickened, with circular bands, and bluish red in color. This hypertrophy and dilatation extended also proximal to the anastomotic opening for about 9 or 10 cm. Above this the bowel gradually became normal. The large bowel was contracted from disuse, otherwise the abdomen was negative. Following this preliminary exploration I excised the pouch much the same as a simple appendectomy and closed the abdomen in layers.

Post-operative history: Convalescence was uneventful. April 30, abdominal pain had disappeared, but patient suffers from occasional diarrhœa. May 10, condition very good, except for recurrent diarrhœa.

PATHOLOGY OF POUCH

This preserved pouch of bowel is 6.5 cm. long and 3.8 cm. in diameter. It is quite smooth externally except where the mesentery was attached and where fibrous adhesions extend part way around it near its middle, forming a ridge which extends about one third of the way around it and a little obliquely.

The valvulæ conniventes are plainly seen in the lining, in all parts of the lining, even in the closed tip. With a hand lens the villi are also plainly seen. On the surfaces made by sectioning the pouch lengthwise so as to divide it into halves, the musculature is easily seen and followed except at one point in the closed end or tip where the circular coat is heavy.

In microscopic preparations of pieces from the surfaces made by bisecting the pouch lengthwise, that is, from a plane of the entire pouch passing through the middle of its largest dimension, there are relatively few noteworthy changes.

The discontinuity of the circular muscular coat over the closed end is naturally conspicuous. The longitudinal muscular coat here is joined to the tunica propria by a thin, well organized scar, and in some of the sections located near the ending of the circular muscular coat on one side there is a slit 2 to 2.5 mm. long parallel with the lining mucosa, a slit in the inner third of the thickness of the wall at this point lined with columnar epithelium and best explained as a diverticulum of the mucosa or as a bit of the lining which has been incorporated in the scar during healing.

In many places the tunica propria as well as the interstices between and within the muscular coats are the seat of a slight œdema. In the perivascular lymph channels, in perivascular situations where lymph channels are less evident or not at all, in all the microscopic preparations and in all layers of the bowel there are small collections of mononuclear leucocytes and plasma cells.

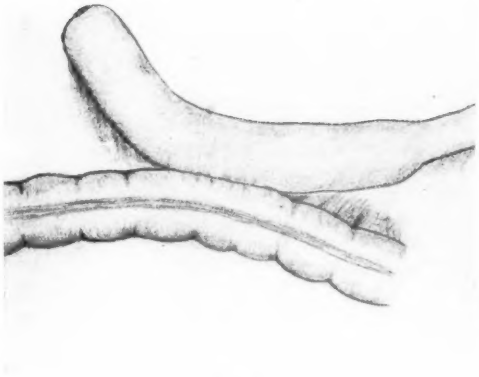


Fig. 1.

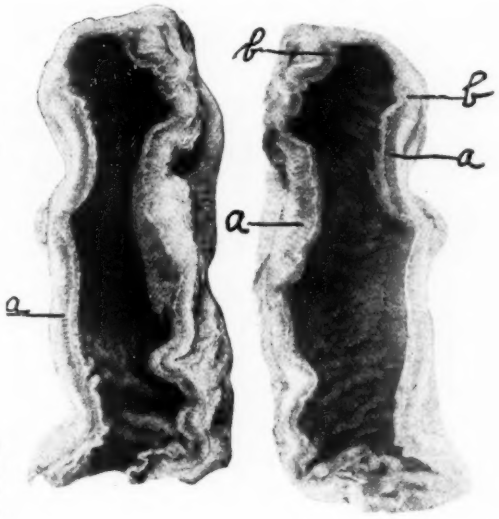


Fig. 2. The pouch cut open lengthwise. The thick circular muscular coat (*a*) is conspicuous. The muscular coat is not continued over the end of the pouch. Its ending is visible at *b*.

These are most abundant in the tunica propria and subserous tissues. They are not in large masses anywhere. In some of them eosinophilous leucocytes are very abundant. There are no alterations in the mucosa.

One of the chief changes is the great thickness of the circular muscular coat. It measures from 1.6 to 1.8 mm. in the different sections.

CONCLUSION

The hypertrophy of the pouch was probably caused by excessive efforts to empty itself of accumulated intestinal contents (or could it possibly be due to conditions brought about by the abnormal iliac outlet with its absent valve and altered environments?).

In performing an ileosigmoidostomy with cut-off ileum the anastomosis should not be made too far from the iliac end as seen from the results in the above case.

It is advisable before performing this formidable operation, especially if it should be done for such a benign condition as constipation, to ascertain the attitude of the patient toward such radical therapeutic measures.

TWO CASES OF OBSTRUCTION OF THE BOWELS FROM UNUSUAL CAUSES[1]

One from Strangulation Caused by a Fibrous Band Across a Hernial Sac; the Other from Intussusception Caused by a Lipoma of the Cæcum

By CLIFFORD U. COLLINS, M. D., Peoria, Illinois
Surgeon to St. Francis' Hospital

THESE cases are not reported because of any interest in the technique of the surgical treatment instituted for the relief of the conditions. The interest lies in the causes of the obstructions and in the diagnostic problems involved.

Case 1. The patient was a male, aged 32, and a barkeeper. On July 26, 1910, at 7:00 P. M., he was taken with vomiting, diarrhœa, and pain through the abdomen. He had had a right inguinal hernia for ten years, and shortly after the attack began he found that the bowel was down in the sac. He tried to reduce the hernia but did not succeed. On July 27 his temperature was 100.5°, pulse 110. He was brought to the St. Francis Hospital, where an effort was made to reduce the hernia, which apparently succeeded. The hernial enlargement disappeared. On July 28 his temperature was 99.5°, pulse 92.

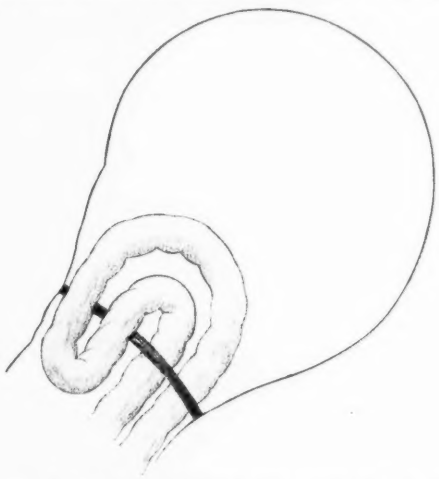


Fig. 1. Diagram of fibrous band across neck of sac with loop of bowel falling over it.

On July 29 he vomited several times and complained of pain in the right lower abdomen. He was tender on pressure over the right lower abdomen. His temperature was 99.5°, pulse 100. His hernia had seemingly been reduced and should not have caused trouble. A diagnosis of appendicitis was made and immediate operation urged.

It was the intention to repair the hernia if the inflammation was confined to the appendix, so the incision was made over the right inguinal canal. After the skin was incised and the fibers of the external oblique aponeurosis separated, the fibers of the internal oblique muscle were separated and the appendix found. Some free fluid escaped from the peritoneal cavity. The appendix was removed and opened, but did not show enough pathological changes to account for all the symptoms. I began to examine the ileum, and about 8 inches from the cæcum I found it firmly attached to the lower right abdominal wall. Further examination revealed that a portion of it was looped over a fibrous band across the internal inguinal ring (Fig. 1). I managed to pass the loop back from around the band. It was discolored, but the circulation was rapidly established after it was released. I succeeded in removing the sac with the fibrous band across the neck, and have preserved it. Uneventful recovery.

Case 2. The patient was a female, aged 50, and married. She had ceased to menstruate two months before I saw her. It had been eight years since her last pregnancy. About two weeks before she came to me she began to feel sick at her stomach and have pain in the right lower abdomen, which had gradually increased. She had vomited several times each day for several days. Her bowels had moved four or five times a day for two weeks. The passages were composed mostly of mucus and were sometimes stained with blood.

There was an oblong mass lying obliquely in the left lower abdomen. This mass was fixed. The uterus was of normal size, and was movable, seeming to have no attachment to the mass in the left lower abdomen. The abdomen was not distended. A rectal examination revealed nothing abnormal. The temperature was normal; pulse 88.

On account of the cessation of menstruation two months before, and the pain, in spite of the fact that the patient was 50 years old, a diagnosis was made of an extra-uterine pregnancy or a possible malignant tumor of the sigmoid or descending colon.

When the abdomen was opened the colon was seen to be considerably enlarged. When the mass in the left lower abdomen was dislodged and delivered, it was seen to be an intussusception. The intussusception was reduced by pressure upon the distal point. After the reduction was effected a solid round tumor, about two inches in diameter, was felt inside of the cæcum. The cæcum was incised and the tumor was removed. It was attached by a pedicle to the inner wall of the cæcum. It proved to be a lipoma. Evidently the tumor had started the intussusception. The patient made an uninterrupted recovery.

In Surgery, Gynecology and Obstetrics for August, 1909, Stetten of New York reports 2 cases of lipoma of the intestines and classifies 75 more cases found in the literature. Of these, 41 were found in the large intestine, of which 6 were found in the cæcum. This indicates the rarity of this condition.

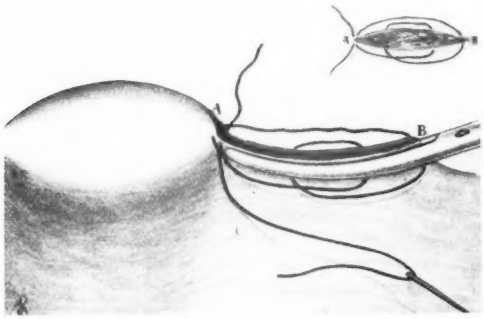
[1] Read before the Western Surgical Association, at Cincinnati, Ohio, December 20, 1912.

MODIFIED FIGURE OF 8 SUTURE [1]

By THOMAS J. WATKINS, M. D., Chicago

THE accompanying illustrations clearly show the features of the suture. We have used this suture in various parts of the body for many years to stop bleeding and to cover broad surfaces of incised tissue. The special advantages of the suture are:

1. The knot is tied over very little tissue; consequently the dangers of the suture breaking while tying, or the knot slipping later, are minimized. These dangers are considerable when a suture is tied over a "long bridge" of tissue.



2. The central portion of tissue cannot retract and bleed because it is included in a distinct loop of the suture.

3. The raw surface is mostly or entirely buried. When the ends of the suture are drawn taut, *which should be done before tying*, the two ends of the cut surface are approximated and bury the denuded tissue. When used on the broad ligament, the central loop includes the ovarian vessels. Any raw surface that may remain is readily covered by a fine "purse string" suture passed superficially through the fundus, the round and infundibulo pelvic ligaments. This suture also draws the fundus forward and prevents backward displacement of the uterus.

The modified figure of 8 suture is useful in doing an appendectomy in cases where the incision through the meso-appendix is so long that there is danger of retraction and hæmorrhage. Raw surface is also covered.

The suture has been found very efficient in the removal of large hemorrhoidal masses. The hemorrhoid is clamped, the suture applied, and as the suture is drawn taut the clamp is removed. In many cases, all o the raw surface is thus entirely buried.

As very little tension is required, fine suture material can be used and thus strangulation of tissue is minimized. Care should be taken to always remove the clamp and draw the suture taut before tying.

[1] Read before the Chicago Gynecological Society, April 18, 1913. See discussion page 510.

TRANSACTIONS OF SOCIETIES

CHICAGO SURGICAL SOCIETY

REGULAR MEETING HELD APRIL 4, 1913, WITH THE PRESIDENT, DR. CHARLES DAVISON, IN THE CHAIR

Dr. Carl G. Swenson read a paper (by invitation) entitled "Tuberculin in Surgical Tuberculosis, with Clinical Reports and Late Results; also Presentation of Cases." (See p. 437)

DISCUSSION

DR. A. J. OCHSNER: Most of the later authors seem to agree that if tuberculin is given properly there is undoubted benefit. My observations go back about nine years, when my brother began to use this treatment in a large number of cases at the Augustana Hospital. At that time my personal feeling toward the treatment was that it was a fad which would probably not stand the test of time. I looked upon it as a kind of cross between Christian Science and high potency homeopathy, and explained my views rather frequently to my brother; but within a year he had proven that his patients did very much better than mine, and from that time on I turned all my tuberculosis patients over to him. At that time he kept two assistants busy all the time taking opsonic indices. I think he established the fact that the reason so many authors gave unfavorable opinions was because they felt the more tuberculin they gave patients the better while it is much more important never to give too much tuberculin. The moment you give too much you do more harm with the treatment than you do good. In other words, you change chronic into acute conditions as Koch did with his original tuberculin. If you use the opsonic index as an aid, carefully and constantly, you can determine the highest amount that you can use, in order to carry the capacity of the leucocytes to their highest point. The moment you go beyond that, you go backwards rather rapidly.

The importance of Dr. Swenson's paper lies in the fact that all of his patients in this series were treated a long time ago, and that all of them are well. There is a distinct advantage in the combined over the surgical treatment. If you treat a patient for tuberculosis surgically only, the chances are that he is almost as tuberculous after you get through with him as he was when you started with him. You have removed a certain circumscribed tuberculous area, but you have not gotten anywhere near curing the patient. The most important part in tuberculosis treatment of every kind is the dietetic and hygienic treatment, no matter what else one does. If patients are treated hygienically and dietetically, they will probably produce enough antitoxins normally to destroy the tubercle bacilli; but if you can study the capacity of the phagocytes by determining the opsonic index and help them along with this method, you can accomplish the same results more rapidly.

As to Dr. Swenson's patients, so far as their general appearance and the condition of the wounds are concerned, they would indicate that they have really recovered fully and permanently.

DR. D. A. K. STEELE: I quite agree with the statements made by Dr. Ochsner. I am probably more radical on account of the longer years of experience I have had with the surgical treatment of tuberculosis, coupled with the dietetic and hygienic treatment, and the limited experience I have had with the use of tuberculin. I have seen so many cures, such rapid and marked improvement under the old method of treatment, with the removal of infected glands, and building up the resistance by proper diet and proper hygienic surroundings, so many patients having recovered and having made just as good recoveries as we have seen in these patients to-night, but still in a number of cases in the last two years it has seemed to me the recovery perhaps was aided somewhat, or the convalescence shortened somewhat, by the addition of the tuberculin treatment.

DR. CHARLES ADAMS: My experience with the use of tuberculin has been exceedingly limited, and I am not in a position to judge of its merits from that experience. I should consider it an exceedingly valuable adjunct to properly applied surgery and hygienic and dietetic means of treatment. I do not think by itself it cuts much of a figure, but it is probably likely to prove a

valuable aid in the future treatment of these cases. I should not want to give up surgery of the neck yet.

DR. FREDERIC A. BESLEY: My experience in the use of tuberculin is limited to the work that Dr. Hollister was good enough to do for me when he was very enthusiastic about the use of this therapeutic agent. He checked up all his work with the opsonic index control, and by this means the dosage was carefully governed. Many of these cases showed a marked improvement, but I was not convinced that other cases of similar character, treated with dietetic and hygienic measures, did not show just as marked improvement.

At the present time, we are not using tuberculin in surgical tuberculosis as routine practice. This position may be a little too arbitrary, for one must grant that in selected cases tuberculin seems to be a beneficial agent.

DR. NORMAN KERR: I should like to relate the experience we are having at the Policlinic Dispensary under the direction of Dr. McMichael. You can readily understand that the condition of dispensary patients is not very favorable. Under the use of old tuberculin for four years they had an average gain in patients of one pound a month. Two years ago, for some reason or other, they changed to the bacillus emulsion, and they have an average gain of a pound and a half a month. Previous to my interest in this dispensary work, like the majority I was rather impatient in waiting for results, and I was disposed to be radical. I do not think, however, tuberculin should be forgotten by any of us. Nobody claims that any of the other measures should be discarded. This is simply a measure to increase the immunity of the patient to the tuberculous process, and it should be carried out very faithfully and for a long period of time.

DR. D. B. PHEMISTER: Certain cases of joint tuberculosis last for years or recur after intervals of apparent healing because of the presence of sequestra in the ends of the bones. These cases should be operated on and the sequestra removed following which tuberculin and other non-operative measures should be employed. It is as irrational not to remove a large piece of dead bone in tuberculosis as in ordinary osteomyelitis. As an example I want to show the X-ray plates and specimen obtained at operation of the following case:

A man 27 years of age had suffered from tuberculosis of the knee for about 10 years and had gone through the usual orthopedic and non-operative treatments without deriving any permanent benefit. The X-ray picture shows, aside from destruction of the joint cartilages and marked regional bone atrophy, a large wedge shaped area in the external condyle with its base upon the joint surface, which casts a very much heavier shadow than the rest of the bone. The joint was resected by Dr. Dean Lewis and in the lower end of the femur which was removed you can see a cone-shaped dense sequestrum corresponding to the location of the shadow in the X-ray plate. The surrounding bone is markedly atrophied as a result of the immobilization and presence of the tubercular process. This patient, 8 months after the operation, is practically cured.

I have seen a number of such cases at the Presbyterian Hospital and they are as distinctly surgical cases as the ordinary osteomyelitis until the sequestra have been removed.

DR. SWENSON (closing the discussion): I have very little more to say other than this: that from the experience I have had, there are cases of tuberculosis in this group that never would have been cured without the use of tuberculin added to other measures. I think tuberculin is a valuable adjunct. Of course, it cannot remove bone sequestra, nor diseased, degenerated, caseous processes, but it is a valuable aid along with other measures. I have had numerous patients get well with it who had previously failed to respond to long continued careful treatment without the aid of this remedy.

CHICAGO GYNECOLOGICAL SOCIETY

REGULAR MEETING HELD APRIL 18, 1913, THE PRESIDENT, DR. RUDOLPH HOLMES, IN THE CHAIR

Dr. Bertha Van Hoosen presented the following cases with specimens:

This specimen is an appendix. A diagnosis of ovarian tumor, in a woman of 60, was made. It had been producing attacks of severe pain and causing violent attacks of vomiting. When first removed it was 5½ inches in circumference and 4½ inches in length. It was entirely cystic. The only interesting thing about it was the extreme size, and the fact that it was dropped into the posterior cul-de-sac, although it was very freely movable. Every time it was moved the woman had violent attacks of vomiting. There was nothing but clear straw-colored fluid in it.

The other specimen is a deciduoma malignum. I first saw the patient in February, 1912. She had been married six years and had two children, one of whom weighed 12 and the other 16 pounds. Both labors terminated by craniotomy. She was, at the time she came to me, two weeks over her menstrual period, and was pregnant. During this pregnancy she was seen by me or Dr. Stine every two weeks for five months; after that every week until she went to the Mary Thompson Hospital. The urine was normal, but she complained of severe pain during the whole nine months. That is the reason we saw her so often. The pain was all through the pelvic region and she vomited during the whole period. On October 8th the labor began and was terminated by Cæsarean section. The measurements of the pelvis made it impossible for her to give birth even with the forceps. The placenta was attached to the anterior wall of the uterus. The child did not breathe for 16 minutes; after that there was no difficulty. Convalescence was normal and the patient was discharged October 25th.

One month later the patient returned to the hospital on account of a continued bloody discharge from the uterus, and remained there until December 24th. Under ergot, hot douches and rest in bed the bleeding entirely ceased and she left the hospital improved, but complaining of feeling weak. The uterus was well involuted and there was no suspicion whatever of any disease of the organ. She nursed the baby all this time, and continued to do so until three weeks ago. January 18th, a month after she left the hospital, she returned again on account of continued bleeding and weakness. She was curetted three days after she entered. The curettings were reported as placental tissue, but without malignancy. The uterus was still normal in size and there was nothing to excite suspicion. The patient left and was not heard from until March 24, 1913, when her husband said she was very sick and thought she was going to die. She was brought to the hospital with a bloody, foul-smelling discharge, with a temperature of 103° F., and respirations of from 30 to 44. The uterus was enlarged to the size of a two-month pregnancy. At the end of 24 hours the patient was curetted and a large amount of foul-smelling grayish tissue, was removed which was determined to be deciduoma malignum. A week later a vaginal hysterectomy with clamps was performed. One ovary had been removed in Europe about two or three years before she was married, so that the remaining ovary and tube with the uterus were removed. The patient steadily improved and is quite convalescent.

Microscopic slides show that there was one place of metastasis in the ovary. I am obliged again to recur to a step interesting to me because it established the fact of a terrific infection. The respirations during the first 24 hours were 36 to 40 or 44, pulse 122 and 120, and this condition continued until after the curettage; about a week afterward the respirations went down but had not been normal until about a week ago. One of the most discouraging things about the case to me was that I should have seen the woman almost constantly for a whole year and should have had the opportunity to have her in the hospital for a month and then should have curetted and had a complete examination and still had to operate on that woman for a deciduoma when *in extremis*. So that the best laboratory methods and the best diagnostic methods do not help us very much in some of these very acute, rapidly malignant cases.

DR. T. J. WATKINS read a paper on "Infantile Type of Uterus with Dysmenorrhœa; Report of Severe Cases Treated During the Last Eight Years." (See p. 460.)

DISCUSSION

DR. H. T. BYFORD: I agree with Dr. Watkins in everything except as to the stem pessary. I tried to use that when I was younger, and after getting a few ulcerations of the cervix and conditions which I did not like I found that I could get along just as well without it. I find that nature has cured these cases partly before we get them. The term infantile uterus is indefinite. They are not infantile uteri. The doctor has said the infantile type. The infantile uterus cannot menstruate. It must be developing to menstruate and by the time it is through menstruating it has got beyond that step of infantilism. Such uteri should be called "puerile," because the functions have been established so that they can menstruate, and of course, the ovarian functions have been established. In the cases of severe dysmenorrhœa which I have treated there is a "puerile" cervix and uterus—as I call it—and when I have sounded the uterus from the outside to the upper part, I find it is 2½ inches and sometimes a little bit more. The body is still small, the junction of the cervix with the body is small and narrow, but during the time since the establishment of menstruation there has been monthly congestion and softening and nature has developed that uterus so that it is no longer infantile.

Dr. Watkins says he does not use the stem to obtain permanent dilatation. Everything else has been done, nearly; the uterus has been developed and I think that is all we want and we cure

these women permanently of their dysmenorrhœa in a large proportion of cases. To illustrate:

A young woman, married a few months, aged 23, menstruating for eight or nine years, was always compelled to go to bed during the period. She commenced with dysmenorrhœa at puberty and always had to have attention and always had to take something, but operative treatment was refused. After she was married, I found the uterus long enough, but there was a very small cervix and a narrow internal os. I put her in the hospital and then dilated very widely and ruptured the external os a little. I stopped and packed thoroughly and for some time afterward. She had but little pain at the next period. I put her under an anæsthetic again and this time succeeded in getting further dilatation, and introduced a packing which I left twenty-four hours—not long enough to cause very much pain—although if the uterus had been small I would have left it until it had caused pain, imitating nature and causing some congestion. She had her next period without any pain whatever and was perfectly well, and never had any pain between the periods. I dilated with sounds frequently—three times a week—in that case, and I found that I was losing a little ground because after a day or two she wouldn't stand it; but after telling her it had to be done she stood it.

Pain with menstruation is largely a subjective mental condition of the patient. Some make a great deal of pain; some have convulsions, as in children; so that I think it is a want of resistance. Of course, they have pain; but pain of great severity, except in inflammation, is not so much due to the dilatation as to the condition of the mind of the patient.

The patient already referred to was kept in the hospital a month and then went back to Detroit with instructions to a gynecologist there from me as to the dilatations. She came back to me in two months with the same pain. The dilatations had not been thoroughly carried out: he had been dilating the uterus but the lower end had not been dilated. I dilated it and the next month she had no pain. This was a few months ago, and this is a typical case. I have patients coming to the office now who have had no pain for many months. I have patients whom I have watched for years, with no pain at all, and the reason is that you keep the cervix dilated long enough to develop the uterus. You overdilate. The point to stimulate the uterus is at the external os, as is illustrated in packing the vagina in an abortion. I have found that dilating the os brings on the menstruation a few days earlier, and keeping on with that you get a permanent relief and a development of the uterus.

One of the first cases that attracted my attention was a patient who had had dysmenorrhœa for years. At first she had no symptoms, but later the symptoms came on, with ovarian pain, and the patient became addicted to morphine. I removed the ovaries in that case for the dysmenorrhœa, which was the first time I ever did that. The ovaries were large and diseased. There were no adhesions.

So, then, I do not see any need for the stem or for anything else. If you want to stimulate the uterus, stimulate the mucous membrane, packing the uterus full and leaving it there for two or three days, although I wouldn't leave it there too long, as you are likely to get round-cell infiltration; but whatever will develop the mucous membrane will develop the undeveloped uterus. If you take out the mucous membrane of the cervix destroyed by a follicular inflammation, the connective scar tissue is absorbed. It is the functionating surface that determines whether the musculature will develop or atrophy. Merely stretching the mucous membrane is enough.

I do not believe in any cutting operations. They immediately stop all attempts to develop the cervix; you will get atrophy rather than development, and of course that may be the lesser of the two evils. If you have a very long cervix and cannot get rid of it you may have to cut it, or if the uterus gets hardened from chronic inflammation you may have to cut; but I mean for developmental purposes cutting is useless. I do not believe you can develop the infantile uterus unless it is done at puberty or soon afterward. After the woman is older you get retrograde changes. If you use too much irritation you are apt to get hyperplasia but not development, and when you get cases of sterility with a small uterus you will find it hard to do anything after 30; but in young persons if you stimulate the uterus by dilatation they will become pregnant. Stimulating the sexual function and gland-secretion is the natural way. The thing that called that to my attention was a case of sarcoma of the ovary in a little girl. The pubic hair began to grow and the breasts developed as a result of the increased congestion of the ovarian tissue. I removed the growth and inside of a year the precocious development had disappeared and the girl grew up and developed normally afterward. Stimulation might be induced by pituitrin and possibly thyroid gland and other remedies to stimulate ovarian congestion rather than to relieve congestion. Certain forms of exercise are useful.

Dr. A. Goldspohn: The line of treatment suggested by Dr. Watkins is something that I have always been afraid of and do not believe in. But the theory on which he applies this foreign body in the uterus to stimulate growth is rational. I think the theory is correct if we could administer this treatment without harm; but there comes the

trouble. The imperfectly developed uterus is a flaccid one, with thin walls, and will not stand up and preserve its proper curve under the impact of the intra-abdominal pressure. It curves upon itself under that pressure. The dysmenorrhœa I would say emphatically is never due to too close a cervix, unless it be in patients who have been the victims of unwise cervical cauterization to excess, or who have been infected so that there has been a deep and pernicious cervicitis excited; or connective tissue hyperplasia may make the cervix too close; but that is usually not present in this type of patients, who are virgins. Therefore, we do not need anything to dilate the cervical canal. This is practically always wide enough to give issue to the normal discharge of menstrual blood. I do not include membraneous dysmenorrhœa: that is a different thing; but we are dealing with normal menstruation making pain for the patient. The normal menstruation or even an excess will have access through the cervical canal. The pain comes because the entire uterus is not sufficient to attend to its function in a woman of that size. Many of these women are well developed physically otherwise, but the uterus is only half the size it ought to be, and therefore the changes which should occur in the circulation of the body of the uterus and in the endometrium of the body proper do not occur, and the general physiological congestion which occurs and prompts menstruation is not relieved and this gives rise to pain. That pain will not be altered by having the cervix long or short or straight or crooked. But often the dysmenorrhœa is simply a mechanical one, not of imperfect development or insufficient function of the uterine body; but this flaccid uterus curves on itself and we have fixed or flexible anteflexion of the uterine body. At first it is flexible. If the condition persists, it becomes more or less fixed and the women of course have mechanical obstruction to the outflow of blood. Now, the object would be to introduce some tube to hold that uterus from bending on itself from intra-abdominal pressure. To introduce a tube with this pressure still on it means an impact at the end of the tube where it comes in contact with the endometrium. This means pressure-atrophy and ulcer.

It occurred to me while the doctor was speaking that we might carry out the idea of a foreign body in the uterus with the idea of stimulating development—a silver tube—if the patient is guarded with regard to hygiene and cleanliness. It is possible such a body could be kept there without getting serious infection if pressure-atrophy from a fixed, inflexible tube could be avoided. A flexible silver tube such as is used by the otologists to drain the mastoid might be introduced without causing objectionable injury to the endometrium. I have for my part not found the need of introducing these things, but I do have to do with the results of this imperfect development in the form of flexible and fixed anteflexions, and therefore I believe, with Dr. Byford, that all operations are irrational, unless to cut through the external os to allow of the exit of a cervical discharge which may accumulate between the internal and external os. I find I get the best results by carefully dilating under full anæsthesia and with all aseptic details, taking plenty of time —never less than half an hour—to dilate such a cervical canal, bearing in mind that any undue stretching with a divulser is harmful to the patient. Such dilatation should be up to the size of a 15, 16 or 18 English sound, and then what curettement may be indicated should be done and this followed with a chemical cleaning out of the interior, then pack the entire uterus from the bottom out with iodoform gauze or some equivalent with plenty of antiseptic in it to keep it sweet for at least three days, if not more, for a special purpose which I shall mention, so that there cannot be any harm from the amount of antiseptic incorporated in the packing, and so that there can arise no harm from infection. Then take particular pains to pack widely through the cervical canal. I get as big a wad of gauze in that constricted portion of the cervical canal as I can possibly get there out to the external os, leaving it there not one day, but three, or possibly four, if there are no contra-indications. The idea is that I want that gauze to be a foreign body there until the uterus shall contract. This foreign body will sulcify the cicatrized, indurated portions of the cervical canal better than any other, and we will get an os that will stand fairly patulous without evil results to the patient. Then continue, in the after-care, to dilate that uterus as Dr. Byford has suggested, very much as the genito-urinary surgeons would dilate a stricture in the male urethra, for a number of months, so as not to permit the return of this vicious flexion. In that manner we can cure these patients fairly well. Now, theoretically, I think it right to dilate with these sounds, never with divulsers in the office, because we cannot be clean enough, but with sounds that have about half the ordinary male curve. In the shops they are called Pratt sounds. If that kind of a sound is introduced and the sinusoidal current applied it ought theoretically to be good. I

have never tried it, but shall, for from its effect on unstriped muscular fiber, and the way it invites contraction of unstriped muscular fiber, it ought to be a good thing.

I would take issue with Dr. Watkins on the modified suture. This is a clever substitute for the Lawson Tait Staffordshire knot, but it does not accomplish any more, that I can see, and all these methods of taking out the tube and ovary that take in the ligamentum infundibulum pelvicum and bring it up to make a stump against the uterus, in the manner this does, I consider very objectionable, because you are tying the fundus down too low. We who believe that the uterus has a normal position in the body and that retroversion is an evil do not want the uterus tied down in the retroverted position in which that technique would place it. After doing such work on one or both sides I would look out for the position of the uterus, and the nearer I can get it up to the abdominal wall the more comfortable the patient will be; so that Dr. Barrett and I will get that uterus up somewhere near the abdominal wall before we get out. If you could in any manner get the uterus forward after Dr. Watkins' operation you would be putting too much strain on the pelvic ligaments. It also does not extirpate the intra-uterine portion of the tube. We know that tying a string around the tube does not close it permanently. The ligature will be absorbed before the mucous membrane within is absorbed, and if you use a non-absorbable ligature it will cut through the tube by pressure-atrophy and the mucous membrane lining the same will be cut and that tube will communicate with the peritoneal cavity, and will be closed up finally by small attacks of peritonitis. They may close in the majority of cases, but the liability of endometritis and a leak into the peritoneal cavity is imminent, and when it does that it will probably seal the tube. This matter I regard as of sufficient importance to require removal of the tube in its intra-uterine portion by a lemon-slice incision. If that causes bleeding, place a hæmostatic stitch, with a ligature at about the junction of the vessels coming from the uterine and ovarian arteries; then with continuous sutures you can close the section, and then continue down along the side of the uterus with the hæmostatic stitch, and leave the pedicle not up against the uterus, but down at the stump of the infundibulo-pelvic ligament, and there is nothing to draw the uterus down or prevent its being brought forward in what I call the normal position.

Dr. R. T. Gillmore: With regard to flexion and the use of tents in these cases of dysmenorrhœa, I am glad Dr. Watkins called attention to the fact that they are sometimes difficult to remove. I had such a case three or four months ago in which I introduced a tent. I tried to remove it in about thirty-six hours and found it was practically impossible. The tent continued to break off, and before I could remove it I had to cut up the cervix on both sides pretty nearly to the internal os. At last I was able to get the tent out. The idea of using two tents is, I think, a very good one. There is no doubt that these cases of dysmenorrhœa are cured in a certain percentage of cases by the use of the tent or the stem pessary. We know as a matter of fact that these cases are cured by pregnancy better than by artificial means, such as tents and stem pessaries, as Dr. Watkins suggests.

Another thing that I think should be mentioned is the question of tents and dilatation of the cervix in sterility in which the patient has not become pregnant after being married for a time. The cervix has frequently to be dilated. Dr. Dudley did an operation in 1891 which was an evolution from the operation of Simpson, back in the 40's, where the posterior lip is split, especially in those cases of anteflexion of such type that there may be a closure of the internal os. Dr. Simpson's operation was practically an operation for laceration of the cervix, cutting up on both sides and opening the external os in that way. I believe Sims cut up on the posterior lip. Dr. Dudley in 1891 devised an operation that was very ingenious and also rather effective in some cases. I have used it several times, cutting up on the posterior lip in this manner and then making an incision similar to that (indicating), and then brought over the lower portion, bringing the external portion from that angle to this angle (indicating) allowing better drainage and increasing the possibility of conception on account of the straightening of the cervix. This also is effective in dysmenorrhœa, as Dr. Brickner in New York had about 172 cases with 17 per cent of cures for dysmenorrhœa and some for sterility.

Dr. C. Banga: I was interested in what the doctor just said about the details of the operation. It shows that there is something in knowing what passed some years ago. In 1893, the year of the World's Fair, Professor Winckel was in Chicago and came to the Michael Reese Hospital when there was a woman there whom I had taken to the hospital on account of a very profuse discharge and a laceration. She was a patient who

had been operated on by Dr. Dudley. He had split the posterior lip of the cervix up to the neck of an anteflexion to relieve the woman of sterility. She had been married about four years. When I first saw the patient I thought she had had a child, but she told me about the operation, and it seemed to me that the profuse leucorrhœa and the inversion of the cervical canal laterally—it was like a stellate tear—was the cause of her not conceiving. Dr. Winckel saw the patient, too, and thinking the leucorrhœa was the cause of the sterility, advised that I correct the effect of the operation that Dudley did, and I did so and the woman afterward conceived and has had several children since. The splitting of the posterior lip of the uterus up to the angle in that one case did not remove the sterility.

As to the operation for dysmenorrhœa, in those cases having what is described as a pinhole os we usually find retention of the normal uterine secretion which in the course of time causes dilatation of the cervical canal, and I think the sterility in these cases is chiefly due to the blocking of the entrance into the uterus by that mucus. I am in the habit of treating all such cases by a plastic discission. I make a transverse discission, much the same as Simpson did, and then cut out posteriorly and anteriorly a wedge-shaped piece of tissue from the end of the cervix. Then, after having made the transverse cut, I carry the knife in a crescent-shaped line along what I want to be the anterior lip, for instance. Then I go in a wider outer circle so that I get, then, this wedge-shaped piece. When I have taken it out, that lip becomes movable, and by stitching it to the outer line anteriorly and posteriorly I get a gaping opening. In this gap you will see the mucosa of the cervical canal, which of course will remain as mucosa and will not cicatrize. The lateral cut into the cercival tissue I leave untouched. Immediately after the operation there is quite a crater visible, and after the lateral cut which has been left to granulate closes there is always a wide opening present. Invariably after that operation, if there is really a pinhole os which was the cause of the operation, there is much relief during the menstruation, and I have had quite a number of cases in married women in whom conception followed, when previously they had been sterile. In the case of a woman married three or four years, who had had difficult menstruation as a girl, and who did not become pregnant, I examined her and found quite a narrow os, and I did that operation last September, and since February of this year she is pregnant, and I attribute it to the operation. At any rate, the patient believed so, for since the operation she has had quite an easy time during her menstruation.

As to opening up by dilatation with sounds, I think it works very well for the time being, but in order to have any reliable report on such cases it would be necessary to watch them for years. Only then would it be possible to determine whether there was a cure. If done every month the menstruation will be easier, but the problem is to cure these patients so that they do not need medical treatment every month.

Dr. C. W. Barrett: Early in my practice I came in contact with Dr. Newman's work. Although it is said that cutting operations are contra-indicated, I have never seen any work that met the necessities of the case in so many instances as Dr. Newman's plastic amputation did. But there was, once in a while, a patient who resisted that treatment. A patient who has had a fixed anteflexion and a great deal of constriction at the internal os is not reached by these measures. I gave considerable thought to this fact, and I think the society will pardon me if I say that I tried to present it to the society and it refused to have such presentation, and I see no particular reason why I should force it on them now; but there are some points that I would like to bring out. In cases of anteflexion, as pictured by Dr. Gillmore on the board, we may sometimes relieve that anteflexion considerably by shortening the round ligaments. These cases of infantile uteri with anteflexion have not only the anteflexion, but have some retroversion as well. In fact, the condition is aggravated by the uterus settling back, the cervix becoming fixed and the retroversion taking on quite a pathological character. Quite a number of these will be improved by shortening the round ligaments and shortening the long cervix which presents in the vagina, the cervix often being 1½ or 2 inches long. A good many operations depend on the proposition that the trouble is due to an external pinhole os. As a matter of fact, the sound will very frequently pass the external os and meet marked resistance at the internal os, and it is the overcoming of the resistance at the internal os that causes the pain in menstruation. Some believe that the clotting of the blood at the internal os causes spasm, as we find in obstruction of the intestine. Now Terrier proposed to overcome that by taking out a V-shaped piece of the cervix. He cautions against hæmorrhage and against the constriction which may follow. Figuring that this trouble was due not only to anteflexion but to stenosis of the internal os, I

proposed at that time to do the operation of cutting the uterus longitudinally a certain distance, separating the edges and sewing it up transversely, which would broaden the posterior wall the required distance and straighten it. I have never seen a uterus so markedly anteflexed that it could not be straightened or turned back in that way. At the same time it enlarges the circumference. That is not the operation for every case of infantile uterus; it is for the severe cases, those that resist cutting the internal orifice, amputation, dilatation, etc. Anything that will cure these patients that require morphine we cannot count irrational. I have never had a case in which that operation was performed that continued to have painful menstruation. Only within the past few weeks I had such a case, in which the painful menstruation ceased after the operation.

DR. N. SPROAT HEANEY: It is a matter of common experience that infantile uteri associated with dysmenorrhœa are best cured by the occurrence of pregnancy. One is led, for a time, to adopt one theory as to the proper operation and at another time another; but the crux of the whole situation, it seems to me, is how does the pregnancy cure the patient? In closing the discussion I would like to have Dr. Watkins tell, for my own instruction, what results are produced by the pregnancy and dilatation so that the patient is cured. Is it the hypertrophy of the uterus as a whole, or is it the tearing of the lower uterine segment and internal os also which results in the production of this, the best, cure? If we could once establish what this pregnancy does, we would perhaps be able to imitate it artificially for the cure of these patients.

DR. F. H. MARTIN: I think there is a little prejudice on the part of those of us who practiced gynecology in the early days of asepsis, or even back in the antiseptic and preantiseptic period, against the stem pessary. Of course the objections we had to the stem pessary at that time would not apply to the work Doctor Watkins would do at this time. But as that old prejudice has clung to me, I would like to have him be a little more elementary as to just how he does the operation, in regard to the precautions he takes in the preparation of the patient, and the method of placing the tube, the preliminary dilatation, if necessary, and other details.

I think the classification is a little misleading too. I think the infantile uterus is purely a tubular uterus, and that sort of uterus, a real infantile uterus, is a very flexible, thoroughly dilatable tube, and not a uterus definitely flexed at the internal os that is difficult of replacement, or, a uterus, in other words, not perfectly pliable. The other forms of uteri, the one with the long cervix and short fundus and the spot position described by Dr. Barrett, are more the uteri of non-development. It is a manifestation of conditions in the pelvis that Dr. Barrett has dwelt on a number of times, and is a matter not as thoroughly taken into consideration in these cases as it should be.

I would like to emphasize a point made by Dr. Watkins himself. Dysmenorrhœa is a local manifestation of a general condition more often than we think. A great many of these cases will, without treatment at all, improve with a change of location. A western girl sent to an eastern school who may have been for a long time the subject of dysmenorrhœa, will immediately improve, and vice versa. A trip to Europe will cure dysmenorrhœa, and I think sometimes the psychologic effect of an operation is to a certain extent the cause of the cure. Women as a rule will not have dysmenorrhœa the first six months or year of their menstruation. I think when the pain appears it is an indication of some lack of balance in the development of the menstrual organs, and it is a question whether it is not a derivative effect of a highly specialized education. One girl will have dysmenorrhœa who is of the monkish type—who is intellectually developed; another who is of the athletic build and who is specializing in athletic studies. The girl who is really healthy is the girl who develops symmetrically; she does society; she is fond of the society of men, and she is developing her sexual side at the same time she is developing her mind and her body. In other words, it is a question whether we are not overeducating our women athletically and mentally to the extent that we are interfering with their being good mothers. That would not do to say at a women's club, but it is a question whether that is not true. So, whenever we discuss this subject, we discuss it just as we do displacements of the uterus—in a circle—and we will keep on discussing it until some of these young men who are getting into the societies will break new ground and take it for granted that everything has not been told and will redevelop gynecology, or we are not going to get very far with the discussion.

DR. REED: Dr. Martin rather anticipated the remarks I was desirous of making in regard to the nomenclature. He covered the ground so thoroughly that there is no use to add to that part of the discussion further than to suggest the term

"immature uterus" to be applied to those peculiarities of formation rather than "puerile" or "infantile," neither of which is the proper descriptive adjective for the condition we find.

The second point is in regard to Dr. Watkins's stem pessary, which it seems to me would be improved by the addition of a stilus with a round point such as the genito-urinary men use in their instrument for insertion into the male bladder, which would allow of easy entrance.

DR. WATKINS: What impresses me most in the paper and in the discussion is that there is so much about this condition that we do not know and which must be learned in regard to the logical treatment of it. Dr. Byford and Dr. Goldspohn agree with me in that it is a question of development, and the logical treatment is a matter of development, and we differ only in the means used to promote this. I believe there is much less objection to the use of the silver tube than there is to gauze packing, and the silver tube is much more efficient because it can be left much longer. It is cleaner. Gauze always decomposes and there is more danger of infection. Every one who has packed a uterus, even a puerperal uterus, must be impressed that it is a difficult procedure to do thoroughly and accurately. Most cases of packing of the uterus is simply plugging of the cervix.

The importance of development is shown in the improvement after marriage where the pelvic organs develop. That would also help to answer Dr. Heaney's question as to how pregnancy and labor tends to improve the dysmenorrhœa. It is more a question of development than the effect on the flexion of the uterus or the small cervix. After labor the dysmenorrhœa is just as well relieved in women who do not get lacerations of the cervix as in those who do. One very often finds a narrower cervix after labor than before the pregnancy took place.

Dr. Byford mentioned that the uterus could not be expected to develop late in life, yet in fibroids and pregnancy we see a great increase in the muscular tissue.

We rather expected that the question of flexion would come up. The amount or its presence seems to have no relation to the dysmenorrhœa. One often sees women in whom the canal of the uterus is parallel with the vaginal canal, but they are well. One sees cases in which the cervix points in the direction of the vaginal canal and the vagina and uterus are about parallel, and the woman is essentially well. One often sees dysmenorrhœa without flexion.

Some of the gentlemen have spoken of the cutting operation. We have used the cutting operation some; incised the posterior lip some and made bilateral incisions. They were good, but chiefly for the reason that the trouble seemed to be at the internal os. There is the point of sensitiveness, as well as there is where the point of constriction is; and it has seemed to me very difficult to make any plastic operation that would extend above the cervix that one could keep open, and such incision is more liable to produce scar tissue than to leave a large canal. However, I feel some hesitancy in criticising the plastic operations on the cervix, as I have not done many.

I believe the question of infection is well covered by two points. One is to be aseptic in the use of the tube, and the second is not to use it in infected cases. The greater danger is in the infected cases. One is very apt to overlook pathogenic bacteria where least expected, and I believe that is an objection to this tube. I have been more fearful, however, of allowing the patient to go out of the hospital with the tube than of infection. A patient could disappear and a malpractice suit could easily result.

As to Dr. Goldspohn's criticism of the modified suture, I would feel sorry if any member of this society would incompletely remove a diseased Fallopian tube. The illustration, however, is possibly a little deceptive and should show some interstitial portion of the tube removed. I believe every member of the society would feel that all of the diseased interstitial portion of the tube should be removed. The criticism as to the tying down of the uterus with that suture simply means that the doctor has not used that suture. In ten years I have not observed that objection.

In reply to Dr. Martin's question about the technique used, we use the general aseptic technique ordinarily used in plastic vaginal operations. Everything is boiled, including the tubes, and no douches are used afterward. The case is clean and we believe will remain much cleaner without douches than with them. In such cases, douches are much more apt to contaminate than keep clean. The nomenclature as mentioned by Dr. Martin and Dr. Reed is very apropos.